# FACTORS CONTRIBUTING TO SMALL INTESTINE SUTURE LINE DISRUPTION

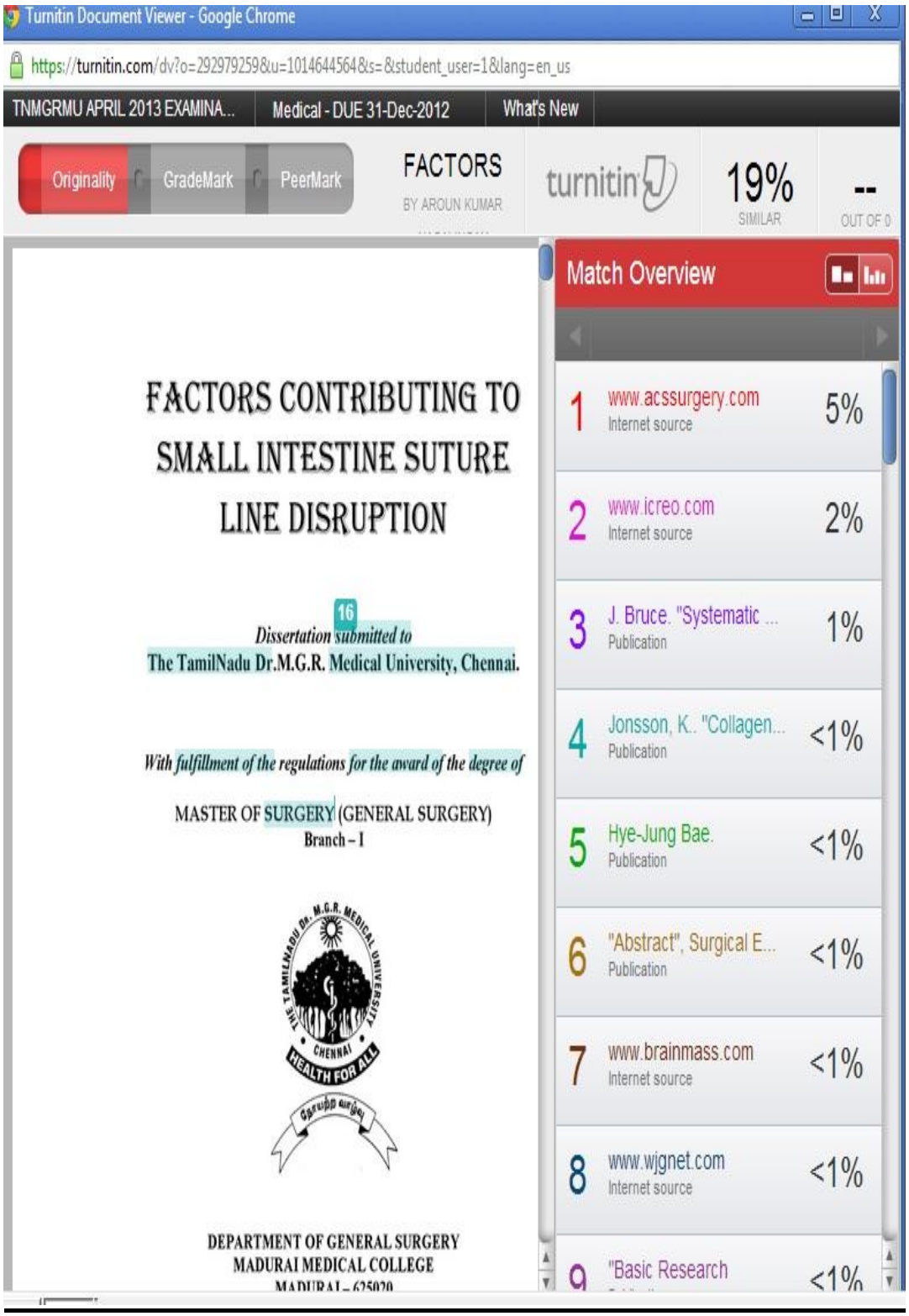
*Dissertation submitted to*
**The TamilNadu Dr.M.G.R. Medical University, Chennai.**

*With fulfillment of the regulations for the award of the degree of*

**MASTER OF SURGERY (GENERAL SURGERY)**
**Branch – I**

**DEPARTMENT OF GENERAL SURGERY**
**MADURAI MEDICAL COLLEGE**
**MADURAI – 625020**

**APRIL- 2013**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# DECLARATION

I , **Dr.N.Arounkumar**, hereby declare that, I carried out this work on **"Factors Contributing to Small Intestine Suture Line Disruption"** at the Department of Surgery, Government Rajaji Hospital, Madurai, under the guidance of **Prof.Dr.D.SOUNDARARAJAN, M.S.,** Professor of Surgery, during the period of November 2011 to October 2012. I also declare that this bonafide work has not been submitted in part or full by me or any others for any award , degree or diploma to any other University or Board either in India or Abroad.

This is submitted to the Tamilnadu Dr.M.G.R.Medical University, Chennai in partial fulfilment of the rules and regulations for the M.S. Degree Examination in General Surgery (Branch I) to be held in April 2013 .

**(DR.N.AROUNKUMAR)**

Place : Madurai

Date :

# CERTIFICATE

Certified that this dissertation is the bonafide work of **Dr. N.Arounkumar** on **"Factors Contributing to Small Intestine Suture Line Disruption"** during his **M.S. (General Surgery)** course from JUNE 2009 to APRIL 2013 at ***THE GOVERNMENT RAJAJI HOSPITAL AND MADURAI MEDICAL COLLEGE, MADURAI.***

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I wish to thank **Prof. SWAMINATHAN M.S.,** Superintendent, Government Rajaji Hospital and Madurai Medical College, Madurai, for his guidance and help to do this work.

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Lastly, I thank **MY PATIENTS** not only for their consent and cooperation towards the preparation of this study but also for the privilege of practicing our craft.

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# INTRODUCTION

The creation of a joint between the bowel ends in surgery is an important practice for a surgeon. This procedure was often performed by a junior surgeon in the emergency setting. There is a little difference between the outcome of this procedure done by a junior surgeon and done by an established surgeon.

To minimize the complications there are few principles which should be adhered during the procedure. For example, the most important thing that should be kept in mind is the presence of excellent blood supply between the bowel edges.

The frequency of anastomotic dehiscence was found to be 1 – 24%. This frequency is high for elective rectal anastomoses than for colonic anastomoses. The postoperative leaks leads to various complications like peritonitis, sepsis, further surgery and the need for stomas.

This prospective study studies the various factors predicting the anastomotic leakage in small bowel anastomoses. These includes patient factors like age, nutritional status etc; Intraoperative factors like the duration of surgery, the nature of the disease, usage of fluids etc., and including the experience of the surgeon. Postoperative factors like need for ventilator and vasopressor support etc.

# AIMS AND OBJECTIVES

- To ascertain the contribution of suture line disruption to post operative morbidity and mortality in patients undergoing small intestinal surgery.
- To identify patients who will be at a higher risk of suture line disruption following small bowel surgery.
- To identify the relative importance of various risk factors contributing to suture line disruption following small intestinal surgery.
- To recommend management strategies for prevention of suture line disruption in high risk groups.

# REVIEW OF LITERATURE

An anastomosis is an artificially created connection between two hollow structures or organs. It has always been an integral part of the science of surgery, a fact apparent from its diverse use across all surgical fields. Gastrointestinal anastomoses have been used with the purpose of joining healthy lumina after resection or bypass of a pathological process occurring in its vicinity. The implications of a failed anastomosis are great, be it in terms of patient morbidity or death. Hence an understanding of the conditions under which its construction can prove dangerous is essential to the surgeon.

## Historical aspects

The earlier mention of intestinal repair is in the Susrutasmhita, a manuscript written by the ancient Indian surgeon Susruta, which dates back to around 6 B.C. Eviscerated intestine resulting from penetrating abdominal injury was examined carefully, washed with milk, lubricated with ghee (clarified butter), honey and returned to the abdomen. Perforations and rents of the gut were closed by the application of black ants before replacing the bowel[1]. His advice concerning the management of cases of small intestinal obstruction was also along similar lines, advocating incision of the gut and removal of any concretions or foreign

bodies. Hippocrates (460-370 B.C) and his contemporary Galen considered small intestinal wounds as uniformly incurable and lethal. The Persian physician, Muhammad Ibn Zakariya al-Razi (860-94 A.D.; alias Razi or Rhazes), used fine strings used by harpists, derived from sheep gut, to suture wounds of the intestine[2]. The first successful small bowel anastomosis was performed by Dieffenbach in 1836, using the technique placed forth by Lembert on the basis of his experimental studies[3]. Most of the advances in the field have been made by numberous European and American surgeons over the last five centuries. They have provided us with the basis of intestinal surgery as stands today.

## Techniques of bowel anastomoses

Entero-enteric anastomoses have been constructed in a variety of manners. The traditional two layer inverted anastomosis has been the most popular, and involves the placement of an inner full thickness absorbable suture, followed by an outer sero- muscular (Lembert) interrupted layer.

Anastomotic techniques have been compared in several studies, and these have revealed that no significant differences exist between the various methods of constructions, perhaps with the exception of everted anastomoses. Goligher and co workers[4] conducted a prospective human trial comparing everting with inverting sutures in 70 cases of large bowel

anastomoses. An unacceptable rate of faecal fistulation was noted in the former group (43% Vs. 8.6%), although the difference in mortality between the groups was not significant (3 Vs. 1). They concluded that everted anastomoses had no place in clinical practice.

Single layer continuous and two layer interrupted anastomoses were compared with each other in a prospective randomized trial conducted by Burch and colleagues, involving a total of 132 anastomoses[3]. Cases involing the stomach, duodenum and rectum were excluded from the study. No significant differences were observed between the groups with respect to anastomotic leaks, abscesses, length of hospital stay. However a significant decrease in operating time and suture material cost was evident with the single layer continuous group.

Waninger et al[5] suggested that the distance between individual sutures be less than the length of the suture from the wound edge, as this would aid in the creation of a moderate suture tension. The latter was experimentally shown by them to provide the best healing pattern at the anastomotic site.

The type of suture material used in the construction of an anastomosis is not of much importance. The inner layer is usually made using a 4-0 or 3-0 absorbable suture such as chromic catgut. As the anastomotic site attains its maximal strength within the first 6 weeks[6], there is little use in placing sutures which will remain beyond that period.

The outer seomuscular layers frequently prepared using 4-0 or 3-0 braided silk, or polypropylene, although the latter has a risk of cutting through tissue after placement.

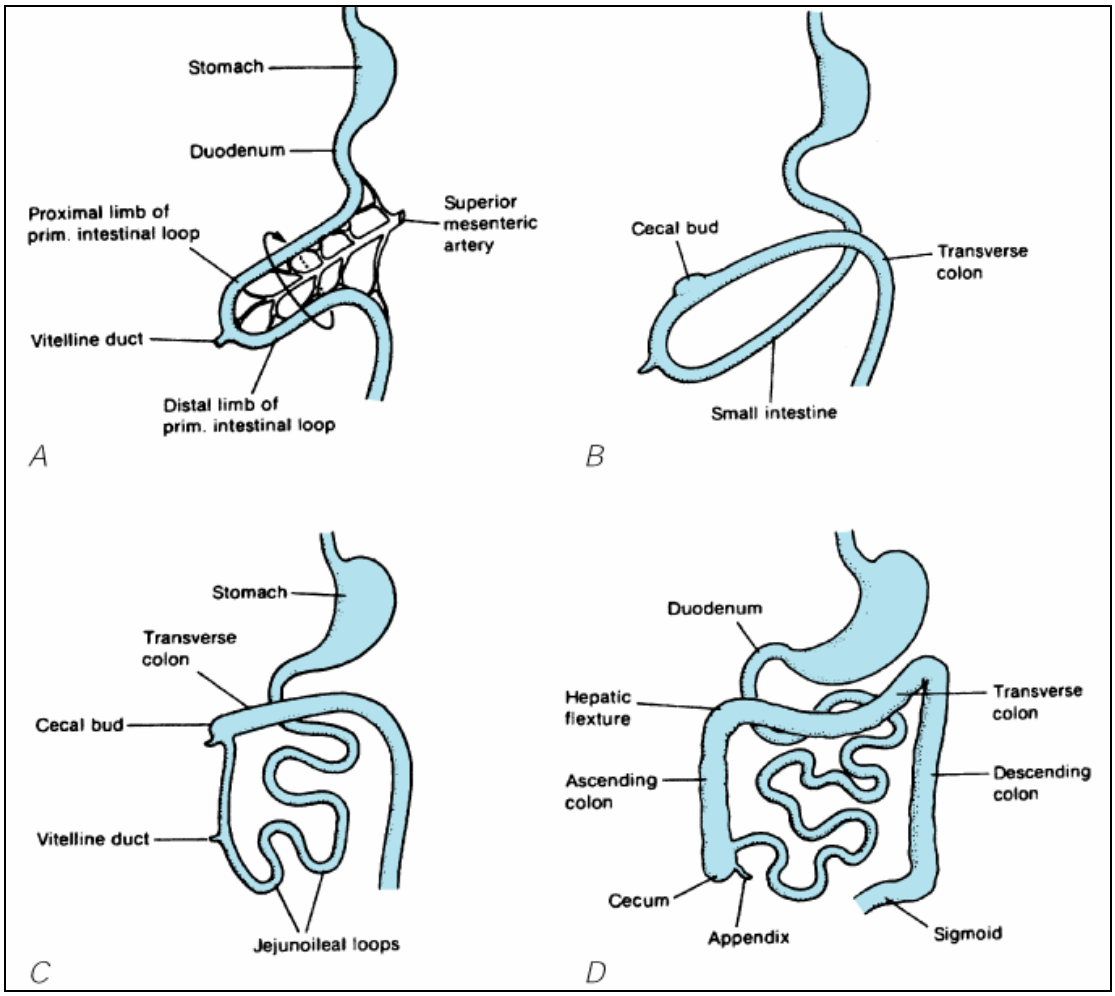
The advent of the stapler has brought about much reduction in the time needed for the creation of an anastomosis, although perhaps having also taken away the element of surgical skill that is required to assemble a hand sutured one. Nevertheless, stapled and hand sutured anastomoses have been shown to be equally reliable[7].

Thus, to conclude, we can say that most of these factors have become part of individual surgeon discretion, and the preference of one method over the other does not alter the ultimate outcome of the procedure.

## EMBRYOLOGY OF SMALL INTESTINE

During the early stage of development, the primitive gut is in free communication with the rest of the yolk sac. In the cephalic and caudal parts of the embryo the primitive gut forms a blind ending tube the foregut and the hindgut and the middle part, the midgut remains temporarily connoted to the yolk sac. In the 5th week embryo, there will be rapid elongation of the gut and its mesentery resulting in formation of the primary intestinal loop. At its apex, the loop remains in open connection with the yolk sac by way of the narrow vitelline duct. The cephalic limb of the loop develops into the distal part of the duodenum the jejunum and part of the ileum. The caudal limb becomes the lower portion of the ileum, the caecum, the appendix, the ascending colon and the proximal two-thirds of the transverse colon. The hindgut gives raise to distal third of transverse colon, the sigmoid, the rectum and part of anal canal.

***Figure 2: Developmental rotation of the intestine***




## Chronology of Rotation of the Midgut Loop

The loop has a prearterial or proximal segment and post arterial or distal segment. Viewed from the ventral side, the loop undergoes an anticlockwise rotation by 90°, so that it now lies in the horizontal plane. The pre-arterial segment comes to lie on the right side and the post-arterial segment on the left.

- Pre-arterial segment now undergoes great increase in length to form coils of the jejunum and ileum and the loops still lie outside the abdominal cavity to the right side of the distal limb.

- The coils of the jejunum and ileum (pre-arterial) return to the abdominal cavity. The coils of jejunum and ileum pass behind the superior mesenteric artery into the left half of the abdominal cavity.
- The post-arterial segment of the midgut loop returns to the abdominal cavity, it also rotates in an anticlockwise direction so that the transverse colon lies anterior to the superior mesenteric artery and the caecum comes lie on the right side.
- Gradually the caecum descends to the iliac fossa and the ascending, transverse and descending parts of the colon become distinct.

## Fixation of the gut

Small and large intestine are suspended from the posterior abdominal wall by mesentery. After the completion of rotation of the gut, the duodenum, the ascending colon, the descending colon and the rectum become retroperitoneal by fusion of their mesenteries with the posterior abdominal wall. The original mesentery persists as the mesentery of the small intestine, the transverse mesocolon and the pelvic mesocolon.

There are three errors in the stages of rotation.

1. Non-rotation
2. Reversed rotation
3. Malrotation

## Pathological consequences of anomalies of rotation[6]

- No functional disturbance may result from abnormal fixation.
- Deficient fixation causes ptosis, torsion and volvulus.
- Excessive fixation may cause interference with mobility, kinks and compression of intestine.
- Abnormal rotation predisposes to volvulus, which causes intestinal obstruction.
- Volvulus of the ileocaecal segment is the typical lesion in later life resulting from imperfect rotation or deficient fixation of the gut.

## Accessory bands of peritoneum

can cause (1) Intestinal obstruction(2) Kinking (3) Angulation of bowel. Failure of part of the original membrane to disappear, minor alterations in the development of secondary mesentery result in accessory peritoneal bands.

These are :

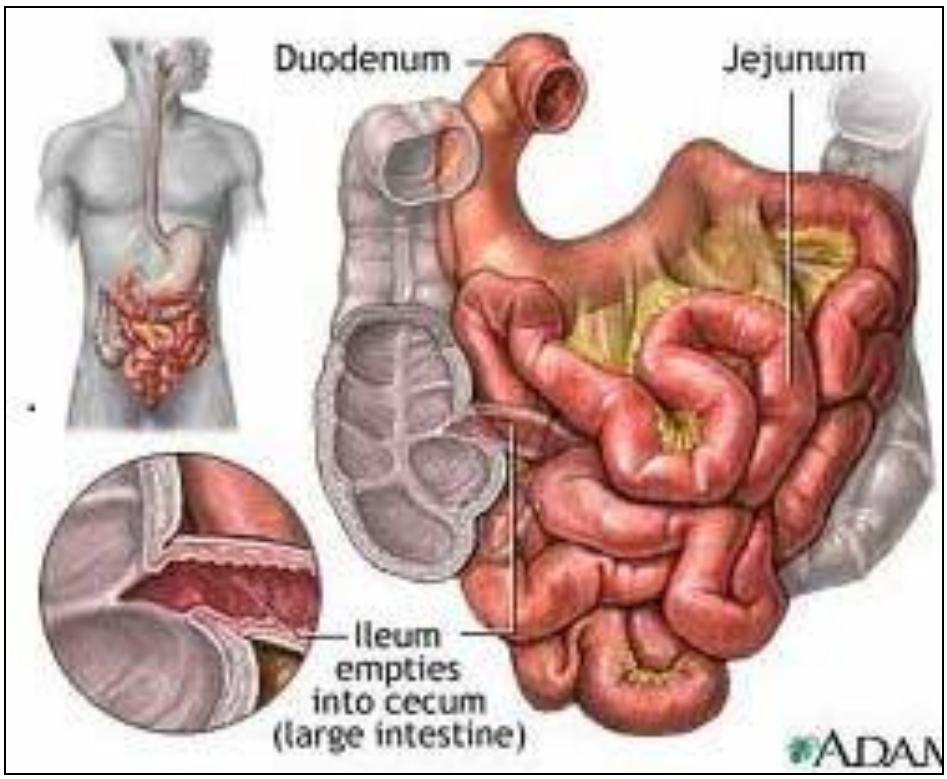
- **Lane's ileal band:** The thickened peritoneal band extending from the right iliac fossa to the 5 cm of ileum which on continuous contraction causes kinking of the small bowel and resulting in obstruction.

- **Mesosigmoid membrane** (Lane's first and last band): This is formed by the thickening of peritoneum extending from the pelvic brim of left iliac region to the junction of descending and sigmoid colon.
- **Genitomesenteric fold of Douglas:** causes kinking of appendix causing obstructive appendicitis as it extends from the back of the terminal mesentery to the region of the suspensory ligament or ovary or testis.
- **Jackson's membrane:** Lies between the posterior abdominal wall and caecum or ascending colon on the right side or from the hepatic flexure to caecum.

## ANATOMY

## SMALL INTESTINE

***Figure 2: Small intestine***




The small intestine is the longest part of the gastrointestinal tract and extends from the pyloric orifice of the stomach to the ileocaecal fold. This hollow tube, which is approximately 6-7 m long with a narrowing diameter from beginning to end, consists of the duodenum, the jejunum and the ileum.[7]

The adult duodenum is 20-25 cm length and the name coined as duodenum because length is as long as width of 12 fingers. It is shortest, widest and most fixed part. It has no mesentery and partially covered by peritoneum. Its course presents a remarkable curves somewhat like horseshoe type, the convexity being directed towards the right and

concavity to the left embracing the head of the pancreas. It has been divided into four portions. First part (superior portion), Second part (descending portion), third part (horizontal portion) and fourth part (ascending portion).

**Blood supply and nerve supply**

Arteries supplying the duodenum arise from the right gastric, supraduodenal, right gastroepiploic, and superior and inferior pancreaticoduodenal arteries.

**Veins:** These end in the splenic, superior mesenteric and portal veins.

**Nerves:** They come from the coeliac plexus.

**Lymph nodes:** Along inferior and posterior pancreatico duodenal artery.

**JEJUNUM AND ILEUM**

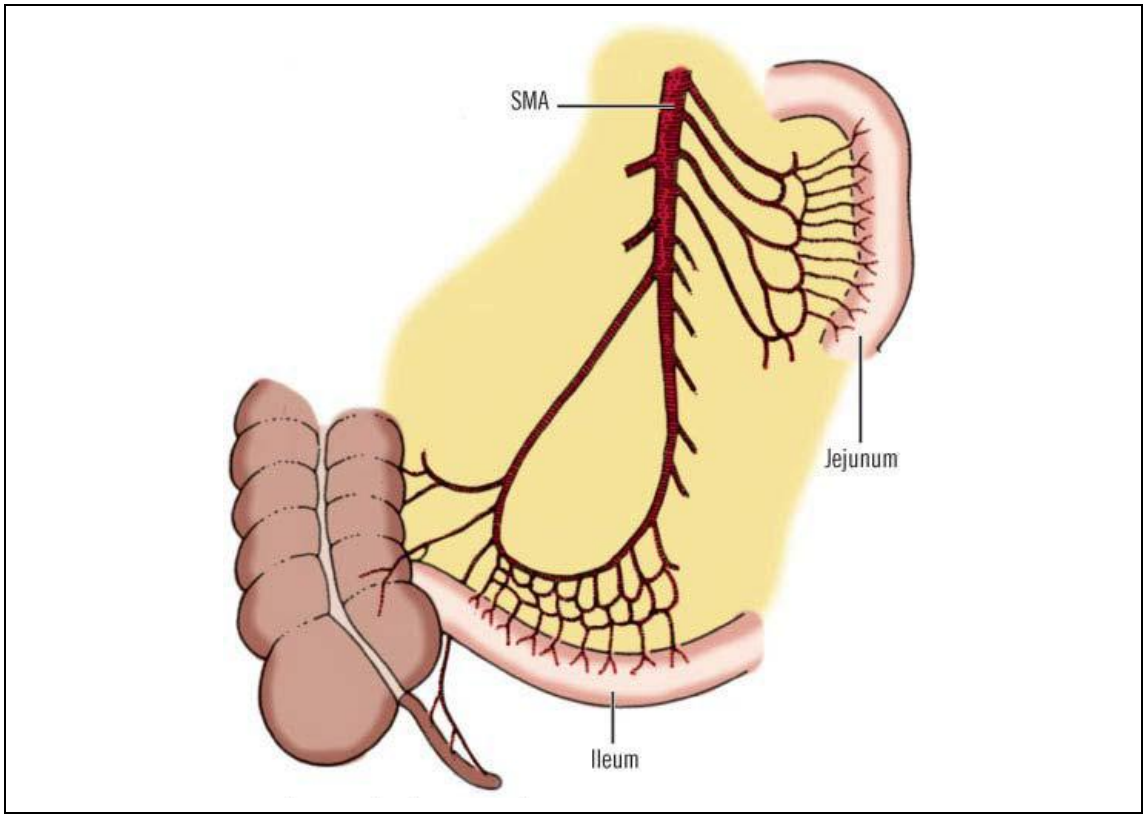
In small intestine excluding duodenum, upper 2/5 are formed by jejunum and lower 3/5 are ileum. The rest of the small intestine extends from the duodenojejunal flexure to the ileocaecal valve, ending at the junction of the caecum and ascending colon. It is totally covered by peritoneum and it is arranged in a series of coils attached to the posterior abdominal wall by the mesentery. The jejunal loops characteristically situated in the upper abdomen to the left of midline, whereas the ileal loops tend to lie in the lower right part of the abdomen and pelvis. This distribution can be reversed during paralytic ileus or small bowel obstruction due to rotation around the mesenteric attachment following bowel distension.

The wall of jejunum and ileum is composed of serosa of visceral peritoneum, muscularis of longitudinal and circular smooth muscle fibres and a mucosa of connective tissue, smooth muscle and epithelium.

## Blood supply

Blood supply is by superior mesenteric artery which is a branch of aorta, the branches of which reaching the mesenteric border extend between the serosal and muscular layers. After this, numerous branches traverse the muscle, supplying it and forming an intricate submucosal plexus from which minute vessels pass to glands and villi. The superior mesenteric veins follow the arteries.

***Figure 3: Blood supply of small intestine***



## Nerve supply

Nerve supply by vagi and thoracic splanchnic nerves through the celiac ganglia and superior mesenteric plexus.

## LARGE INTESTINE

It is about 150 cm long, it extends from the terminal ileum to the anus. Its function is chiefly absorption of fluids and solutes and it differs in structure, size and arrangement from the small intestine in the following ways:

- It is for the most part more fixed in position.
- Its longitudinal muscle, though a complete layer, is concentrated into three longitudinal taeniae coli.
- The colonic wall is puckered into sacculations (haustrations) and appendices epiploicie by the taeniae.
- It has a great caliber.

The divisions are caecum, colon proper and the rectum.

## Caecum

The caecum is a blind pouch lies in the right iliac fossa, its average axial length is about 6 cm and its breadth about 7.5 cm continues proximally with the distal ileum and distally with the ascending colon and is related posteriorly to iliopsoas muscle and femoral nerve, anteriorly to the abdominal wall, greater omentum and the coils of ileum. Almost the

entire posterior surface of caecum is attached to the abdominal wall, in some cases it is wholly unattached.

## Ileocaecal valve

The ileum opens on its posteromedial aspects of the caeco-colic junction and two flaps which project into the lumen of the colon. The valve is actually closed by sympathetic tone. It is mechanically closed by the distensions of caecum and prevents the reflux of caecal contents into the ileum and regulates the flow of ileal contents.

## Colon

The colon is conveniently considered in four parts: (1) Ascending, (2) Transverse, (3) Descending and (4) Sigmoid.

### *Ascending colon*

It is normally fused with the posterior body wall and covered by peritoneum anteriorly. It is about 15 cm long and narrower than the caecum it ascends to the inferior surface of the right lobe of the liver, on which it makes a shallow depression, here it turns abruptly forwards and to the left; at right colic flexure.

### *Hepatic flexure*

Anteriorly covered by peritoneum, posteriorly not covered by peritoneum and is in direct contact with renal fascia. It is related posteriorly to the inferolateral part of the anterior surface of the right

kidney above, and anterolaterally is the right lobe of the liver, anteromedially are the descending part of the duodenum and fundus of the gallbladder.

***Transverse colon***

It extends from the right hepatic flexure to the left colic flexure measuring 50 cm. The transverse colon, unlike ascending and descending colon has a mesentery that had secondarily fused with posterior wall of the omental bursa. The transverse colon hangs in U or V shaped curve. Above the transverse colon are the liver and gallbladder, the greater gastric curvature and the lateral end of spleen, below is the small intestine, in front are the posterior layers of the greater omentum and behind are the descending part of the duodenum, the head of the pancreas, the upper end of the mesentery, duodenojejunal flexure and coils of the jejunum and ileum. The transverse colon sometimes may be interposed between liver and diaphragm (Chilaiditi syndrome).

***Splenic flexure***

This is the junction of the transverse and descending colon in the left hypochondriac region. It is related to the lower part of the spleen and pancreatic tail above and medially with the front of the left kidney. It is attached to diaphragm by phrenico-colic ligament, which lies below the anterolateral pole of the spleen. It lies more superiorly and posteriorly than the hepatic flexure at the level of 10th and 11th ribs.

***Descending colon***

It is about 25 cm long and extends from the splenic flexure to pelvic brim, and in the whole of its course is plastered to the posterior abdominal wall by peritoneum (like ascending colon). The descending colon is smaller in caliber more deeply placed and more frequently covered posteriorly by peritoneum. The descending colon lies on the lumbar fascia and iliac fascia. It ends at the pelvic brim about 5 cm above the inguinal ligament.

***Sigmoid colon***

It is about 40 cm length. Sigmoid colon extends from the descending colon at the pelvic brim to the commencement of the rectum in front of the third piece of the sacrum. The sigmoid mesocolon has an inverted 'V' attachment to the posterior abdominal wall.

## Blood supply

Blood supply is by branches of superior mesenteric artery and inferior mesenteric artery. Superior mesenteric artery supply upto the junction of middle 1/3$^{rd}$ of transverse colon and colon beyond this is supplied by inferior mesenteric artery.

## Nerve supply

Sympathetic to midgut from coeliac ganglion (T1-L1). Parasympathetic from vagus through coeliac plexus. Hindgut portion

receives sympathetic supply from the lumbar sympathetic chain from L1-L2 and parasympathetic from the pelvic splanchnic nerves.

## Rectum

The rectum is 12 cm long and is continuous with the sigmoid colon at S3. The human rectum follows the posterior concavity of the sacrum and shows three lateral curves or flexures that are most prominent when the viscus distended, upper and lower curves convex to the right and a middle curve convex to the left, the lowest part is slightly dilate as the rectal ampulla. It ends 2-3 cm in front and below the tip of the coccyx, turning abruptly downwards and backwards through levator ani muscle to become the anal canal 4 cm from the anal verge.

The mucosa of large intestine differs from small intestine in that it has no villi and consists of simple columnar epithelium which has only absorptive and goblet cells. The longitudinal muscles of muscularis mucosa are thickened to form taenia coli, on contraction leads to formation of haustra necessary for haustral churning.

## Blood supply

Blood supply mainly from the superior rectal artery, with contributions from the middle and inferior rectal and median sacral vessels. Veins correspond to the arteries, but anastomose freely with one another, forming an internal rectal plexus in the submucosa and external rectal plexus outside the muscular wall.

## Nerve supply

The sympathetic is derived by branches from the hypogastric plexus. The parasympathetic supply is from S2 and S3 by the pelvic splanchnic nerves.

## Lymphatic drainage of colon

Lymph from the colon passes through four sets of lymph nodes: (a) Epicolic lymph nodes, lying on the wall of the colon, (b) Paracolic nodes on the medial side of ascending, descending and mesocolic border of transverse and sigmoid colon, (c) Intermediate nodes along the main branches of vessels, (d) Terminal nodes at the origin of SMA and IMA, finally drains to para-aortic nodes.

## PHYSIOLOGY

The gastrointestinal system consists of the gastrointestinal tract and associated glandular organs that produce secretions.

The major physiological functions of gastrointestinal system are to digest food stuffs and absorb nutrient molecules into the blood stream. Mainly the small intestine and large intestinal carries out these functions by motility, secretion digestion and absorption.

Motility refers to the movements that mix and circulate the gastrointestinal contents and propel them along the length of the tract. The contents are usually propelled in the anterograde (forward) direction.

Secretion – refers to the processes by which the glands associated with the small intestine and large intestine release water and substances into the lumen.

Digestion – defined as the processes by which food and large molecules are chemically degraded to produce smaller molecules that can be absorbed along the wall of the intestine.

Absorption refers to the processes by which nutrient molecules are absorbed by cells that live in the intestine and enter the circulation.

## Functional Anatomy of Intestine

Intestinal villi – the villi are minute projections which are called as enterocytes each enterocyte gives rise to hair like projections called microvilli, within each villi called lacteal. The lacteal opens into lymphatic vessels.

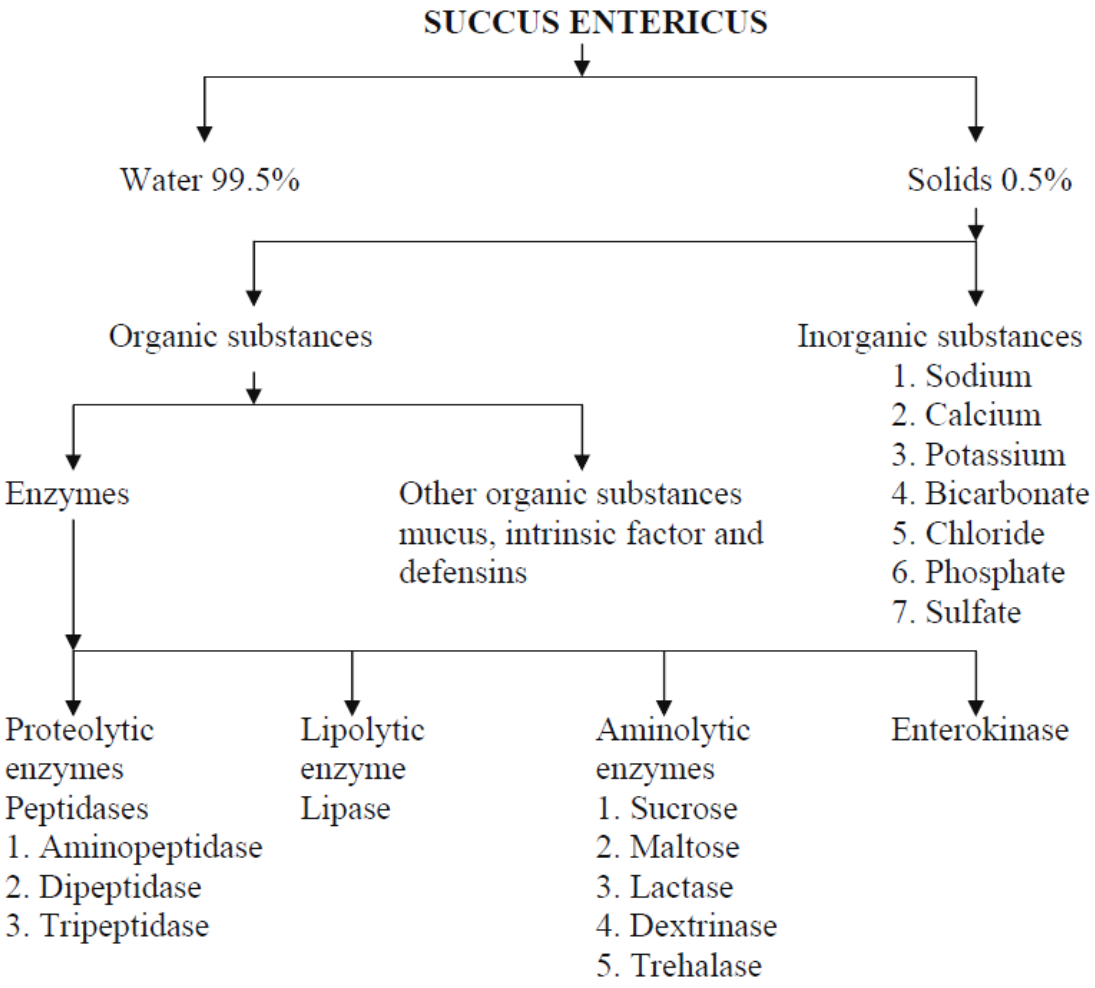
Crypts of Lieberkuhn are simple tubular glands of intestine. The three types of cells are interposed between columnar cells of the glands.

1. Argentaffin cells also known as enterochromaffin cells which secrets intrinsic factor that is essential for the absorption of the vitamin B12.
2. Goblet cells which secrete mucus.
3. Paneth cells which secrete cytokines called defensins.

Brunner's glands: These glands penetrate muscularis mucosa in the first part of the duodenum secretes mucus and traces of enzymes. The small intestine is presented with about 9 litres of fluid per day, 2 litres from dietary sources and 7 litres of gastrointestinal secretions, however only 1-2 litres pass into the colon.

## Properties of succus entericus

Volume – 180 ml/day
Reaction – Alkaline
pH – 8.3
Volume – 180 ml/day




### *Functions of Succus Entericus*

1. Digestive function – The enzymes of succus entericus act on the partially digested food and convert them into final digestive products.
2. Protective function – The mucus present in the succus entericus protects the intestinal wall from the acid chime, which enters the intestine from stomach paneth cells secrete defensins which are the antimicrobial peptides.

3. Activator function – The enterokinase present in intestinal juice activates trypsinogen into trypsin.

4. Haemopoietic function – Intrinsic factor of castle which is present in the intestine, plays important role in erythropoiesis.

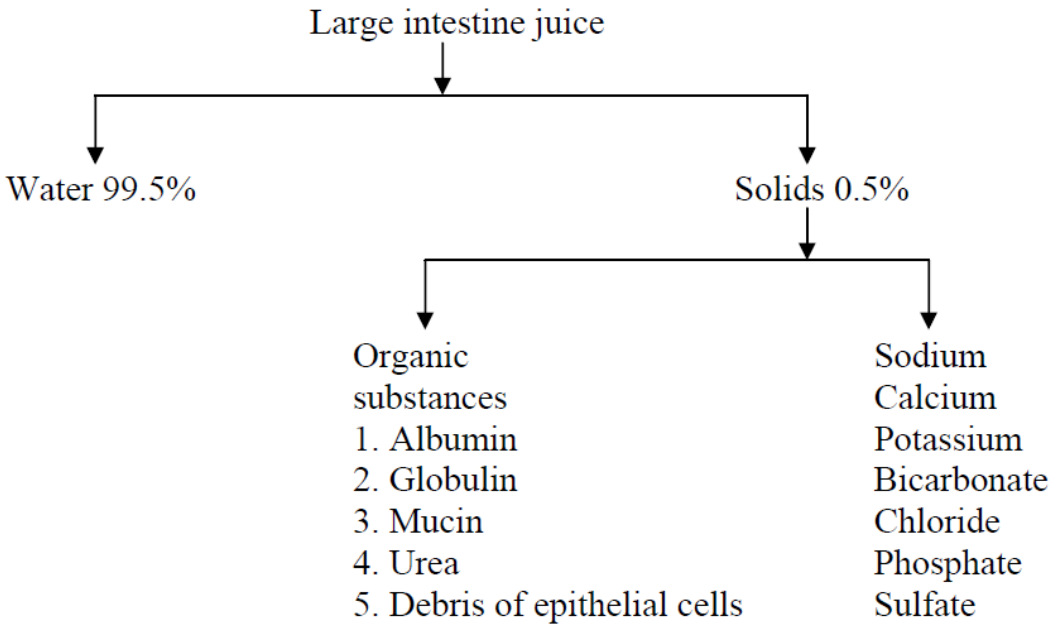
5. Hydrolytic process – Intestinal juice helps in all the enzymatic reactions of digestion.

## Functions of small intestine

1. Mechanical function
2. Secretory function
3. Hormonal function
4. Digestive function
5. Activator function
6. Hemopoietic function
7. Hydrolytic function
8. Absorptive function

## Large Intestine

### *Secretions*




## Functions of large intestine

1. Absorptive function – absorbs various substances such as water, electrolytes, organic substances like glucose, alcohol, drugs like anaesthetic agents, sedatives and steroids.
2. Formation of faeces
3. Excretory function
4. Secretory function
5. Synthetic function – synthesizes folic acid, vitamin B12 and vitamin K

## Movements of small intestine

The movements of small intestine are essentials for mixing the chime with digestive juices, propulsion of food and absorption.

Four stages of movements occur in small intestine.

1. Mixing movements
    a. Segmentation movements
    b. Pendular movements
2. Propulsive movements
    a. Peristaltic movements
    b. Peristaltic rush
3. Peristalsis in fasting – Migrating motor complex
4. Movements of villi

## Movements of large intestine

- Segmentation contractions
- Mass peristalsis

## Intestinal bacteria

The bacteria in the gastrointestinal tract can be divided into three types.

1. Some are pathogen that cause disease.
2. Others are symbionts that benefit the host and vice versa, and most are commensals.

The bacteria include various strains of Escherichia coli and Enterobacter aerogenes. Pleomorphic organisms such as Bacteroides fragilis and cocci of various types.

## ANASTOMOTIC PHYSIOLOGY

The underlying factor in obtaining a good anastomotic result remains correct sub mucosal apposition of the bowel ends. Studies by Jonsson and colleagues[8,9,10] on small bowel anastomotic healing have demonstrated a decrease in the suture holding capacity in the first 3 days post operatively, although no significant change in the collagen content of the intestinal wall was seen. Their animal studies showed an increase in collagen synthesis following small bowel anastomosis not just at the anastomotic site, but also in the other parts of the small gut, stomach and the colon. The immaturity of newly synthesized collagen in the first few days after surgery might account for the quick loss of suture holding capacity in that period. Collagen levels increased in the anastomotic segment predominantly during the phase of fibroplasia (from 4 days post operatively). This localized healing represented actual anastomotic healing, whilst the earlier non specific collagen increase was attributed to the result of operative trauma. 'Cutting through' of sutures was assumed to be a locally mediated process, due to the action of collagenase in the vicinity of the suture, where the presence of proteinase inhibitors was expected to be less due to impaired circulation.

## Definition of anastomotic leak

Anastomotic leak has been previously defined as a leak of luminal contents from a surgical joint between two hollow viscera. The luminal contents may emerge either through the wound or at the drain site, or they may collect near the anastomosis, causing fever, abscess, septicaemia, metabolic disturbance and/or multiple organ failure. The escape of luminal contents from the site of the anastomosis into an adjacent localized area, detected by imaging, in the absence of clinical symptoms and signs should be recorded as a subclinical leak[11]. This definition was subsequently challenged by Bruce et al[12], who noted that it had no anatomical specificity and no chronological cut off point. They proposed new definitions specific to the anatomical site, and also included signs and symptoms, level of severity, and components of clinical management. Leaks were sub classified as radiological, minor clinical and major clinical (those leaks requiring a change in management, or delay in hospital discharge).

## DETERMINANTS OF ANASTOMOTIC FAILURE

### *Local Factors*

Basic surgical principles dictate that certain factors be met to achieve ample healing at the site of a wound, and this applies to an intestinal suture line too. These include a good blood supply at the anastomotic site, proper technique in its construction, an adequate lumen,

and lack of distal obstruction[13]. These factors, which are directly under the control of the surgeon during surgery, should be given the utmost importance lest they be the cause of an anastomotic failure. Nahai and colleagures[14], in their canine experiments, noted that large bowel anastomoses had more of a tendency to leak when compared to their small bowel counterparts, a difference attributed to the effects of spillage of colonic bacteria into the abdominal cavity before or during surgery with subsequent infection of the suture line. Under identical conditions, no difference was noted between small and large bowel healing rates. They also stated that proper serosal coaptation using an inverted suture line, and the use of prophylactic antibiotics reduced the incidence of anastomotic leak.

### *Intra abdominal sepsis*

That intra abdominal infection retards intestinal healing has been established through the demonstration of impaired synthesis of colonic reparative collagen and disordered regulation of collagen gene expression in the face of intra abdominal sepsis[15]. This collagen is essential to the reconstitution of the submucosa, in which lies the mechanical strength of the intestinal wall.

### *Tissue oxygenation*

A good blood supply to the ends of the bowel being anastomosed together is imperative for adequate healing and prevention of anastomotic

disruption. Various methods have been employed in the past to assess the viability of bowel ends intra operatively, including subjective evaluation, intra arterial dye injection and Doppler ultrasound. Sheridan and co workers[16] used a surface electrode to assess the tissue oxygen tension in human colon before and after construction of anastomoses. They encountered a leak rate of 10% in the 50 patients they analyzed. A tissue oxygen tension of less than 20mmHg in the anastomosed region was significantly associated with subsequent disruption, as was a decrease in tissue oxygen tension to below 50% of the pre resection value. They concluded that relative tissue hypoxia was a major determinant of anastomotic leakage.

## Systemic Factors

### *Patient Age*

Much has been said on the adverse effects of advancing age on anastomotic healing, the general belief being that older patients have slower healing tendencies. Earlier studies have cited increasing age as a possible risk factor for intestinal anastomotic dehiscence[17], but experiments conducted by Stoop and others[18] showed that intestinal anastomoses in healthy aged animals healed as well as those in young healthy ones, their conclusions being based on assessments of wound strength and wound collagen content. They presumed that the contrary results obtained in earlier studies were a result of analysis of patients,

rather than healthy human subjects. It was felt that additional, hitherto unidentified factors existed in elderly patients which independently contributed to poor wound anastomotic healing rather than advanced age alone.

### *Abdominal trauma*

The behaviour of repaired intestine in the setting of blunt and penetrating abdominal trauma was analyzed by Behrman and colleagues[19], in their study of 101 small bowel and 66 colonic cases. They encountered a leak rate 8.7% in cases of small bowel resection and anastomosis (4 of 48), although none of the enterrorhaphies in the study leaked (55 of 101). Increasing patient age and large amounts of intra operative blood transfusion and fluid administration were the factors found to have a significant association with breakdown of small intestinal repair. They felt that enteric contamination per se, of the repair site did not play a significant role in the latter's breakdown, as all the cases they had assessed had some degree of such contamination. The investigators have noted that abdominal compartment syndrome occurred in three of the four patients who had a small bowel leak, and have assigned it a cause-effect relationship, having assumed that the compartment syndrome is a direct sequel of anastomotic breakdown, and not vice versa. It is pointed out that splanchnic hypoperfusion, which arises as a part of the

body's general reaction to trauma and hypovolemia, is a major factor that impedes anastomotic healing.

### *Blood transfusions*

The effect of blood transfusion on the immune system has been a matter of debate for over 30 years, from the time it was seen that pre transplant transfusions lengthened allograft survival, to studies demonstrating augmented tumour growth and increased septic complications in patients receiving transfusions in the peri operative period. It was in this backdrop that Tadros et al[20] conducted experimental studies on rats to determine the effects of tranfusions on intestinal anastomoses. They found mortality rates and the incidence of peri anastomotic abscesses to be significantly higher in those animals receiving blood transfusions, as compared to controls that received just crystalloids. Significant reductions in bursting pressures and hydroxyproline content in the region of the anastomoses were found in the transfused group. Wobbes and colleagues[21] did a retrospective analysis on patients who had undergone gastric and colonic surgeries, and found that advanced age, low serum protein (<6g%), and blood transfusions over 3 units to be highly associated with post operative septic complications. However, neither total intra operative blood loss nor duration of surgery was found to have a bearing on the occurrence of the same. Another study noted that the transfusion of more than 2 units

of packed cells intra operatively was associated with a significant rate of leakage[22]. The occurrence of immunosuppression in the setting of blood transfusions has been attributed to blunted cell mediated immunity, decreased helper/suppressor T cell ratios and lowered natural killer cell activity. This could directly translate into impaired anastomotic healing, or on the other hand, lead primarily to an intra peritoneal infection which in turn causes septic complications[20].

### *Malnutrition*

The repercussions of malnutrition are seen in the process of wound healing. Surgical procedure in malnourished patients are associated with much morbidity and a definite risk of mortality as well. Demptsy and colleague defined 'clinically relevant malnutrition' as "…a state of altered nutritional status that adversely affects clinical outome", and further stated that serum albumin, serum transferrin, triceps skin fold thickness and cutaneous direct hypersensitivity tests were the best markers of the state[23]. The prognostic nutritional index (PNI) takes these factors into account when calculating the percentage risk of operative morbidity and mortality. Anthropometic markers have been used to clinically assess the protein stores of the body, but were shown to be of no use in individual patient evaluation, due to the great magnitude of variations encountered[24]. It was found to be reliable only in assessment of groups of patients, although again proving unworthy in following up

these groups for changes in body nitrogen. Brenner et al[25] concluded from their study that upper arm anthropometry and hand grip dynamometry, although being inexpensive and rapid, were unsuitable for identifying patients at high risk of serious post operative complications. Later studies by Detsky and co workers[26] showed that serum albumin and Subjective Global Assessment (SGA) reliably stratified patients undergoing elective gastrointestinal surgery into those that had a high risk of post operative nutrition-associated complications. SGA is based on five features of the patient's history (weight loss in the past 6 months, dietary intake, gastrointestinal symptoms, functional status or energy level and metabolic demands) and four parameters by physical examination (loss of subcutaneous fat, muscle wasting, edema and ascites).

Several studies have established an association between hypoalbuminemia and anastomotic leak[17,22,28,29], whereas others have been unable to verify such a relationship[27,30], Gibbs and co workers[28] remarked that low serum albumin levels were associated with significant increases in operative mortality and morbidity. Hypoalbumineamia was found to be a reliable predictor of sepsis and infection. This finding was corroborated by Kudsk and co workers[29] who found that serum albumin levels below 3.25g/dL were associated with increased post operative complications in patients undergoing elective gastrointestinal surgery. In

addition, they noted that the operative site also influenced the complication rate, esophageal and pancreatic surgeries having a significantly higher morbidity compared to surgeries on the colon, at the same albumin levels. Pickleman and colleagues noted seminal hemoglobin levels in both leak and control groups of their study[30]. Buchmiller-Crair et al[31] concluded that acute anemia (experimentally produced hemodilution) decreased small intestinal anastomotic bursting pressure at two weeks, in a rabbit model. They found decreased leucocytic invasion, decreased angiogenesis and reduced collagen content after histological assessment of the anastomosed intestine. This observations more relevant in the setting of abdominal trauma associated with acute blood loss.

Jex et al[13] found an association between recent weight loss (>10 pounds) and anastomotic complications. Irvin suggested that visceral collagen was less affected by the effects of malnutrition when compared to parietal tissues[32]. His remarks were based on the results of his experimental study on rats, which showed a less profound change in the tensile strength of colonic anastomoses in the setting of malnutrition, in comparison to the decreases in mechanical strength of skin wounds noted in the same group of animals.

## Previous Studies

Earlier studies had elucidated predictors of anastomotic disruption which are now uniformly accepted. Irvin and Golgher, in their retrospective human study, found poor nutritional status, surgeries for malignancy, the lack of, or poor bowel preparation, old age (>60 years), and bowel fixity in the region of an anastomosis, all to be significantly associated with clinical leaks[17].

The vast majority of studies on anastomotic complications have been in relation to the large bowel. Jex and others[13], in their analysis of 519 patients undergoing lower gastrointestinal anastomoses, found a leak rate of 2%. The risk of complications was increased by pre operative variable like colonic obstruction, malnutrition, malignancy and sepsis. Neither the intraoperative variables they analyzed (viz. emergency surgery), technique of construction, suture material, or surgeon) nor factors such as age, sex, steroid use and diabetes mellitus, had a significant bearing on an adverse anastomotic outcome.Preoperative bowel obstruction was noted to be the strongest predictor of such a complication.

Golub et al[22] in their retrospective analysis of 813 anastomoses at most levels of the gastrointestinal tract, found an overall leak rate of 4.5%, and a rate of 9% for anastomoses involving the small bowel. Their multivariate analysis revealed 6 factors to be independently associated

with the risk of an anastomotic dehiscence; namely chronic obstructive pulmonary disease, bowel obstruction, presence of peritonitis, corticosteroid usage, intraoperative transfusion of more than 2 units blood, and a serum albumin level of less than 3.0g%.

Hesp and colleagues[33] in their study of 234 small bowel anastomoses noted anastomotic insufficiency in 17 cases (7.3%), the rate being much higher in the presence of intra abdominal infection (14.8%). They advocated the dismantling of a failed anastomosis and construction of a split enterostomy rather than reanastomosis, as the latter was associated with poorer results.

### *Typhoid Ileal perforations*

Typhoid fever, caused mainly by the gram negative bacillus *Salmonella typhi,* is an important cause of distal small bowel perforations, especially so in developing nations. Most of the deaths due to the disease are on account of its intestinal complications, viz., haemorrhage and perforation[34]. Bowel perforation is usually seen in the second week of the illness[35]. Serologic and bacteriological tests are unreliable, with a high incidence of false negativity. In the presence of supportive history, the intraoperative finding of a terminal lieal perforation along its antimesenteric border is enough evidence for establishing a diagnosis of typhoid fever[36].

Mock and colleagues had a mortality rate of 31% and enterocutaneous fistulation in 5%, in their series of 221 cases of surgically treated typhoid enteric perforation, which used chloramphenicol based drug regimens. They also brought to notice that the mortality rate was increased by the development of an enterocultaneous fistula, and was neither dependent on the type of operation nor antibiotic regimen[36]. Meier et al[34] had a leak rate of 8% in their series of 108 patients. Santillana noted enterocutaneous fistulation in 3 of the 96 patients in his study[35]. Chatterjee and colleagues in their review of surgically treated typhoid ileal perforations over a three decade span involving 351 cases, noted anastomotic leakage in nine patients[37].

## Other determinants of anastomotic leakage

Golub and co workers[22] found chronic obstructive pulmonary disease, bowel obstruction, peritonitis, and corticosteroid usage each to be independently associated with anastomotic dehiscence. Other factors were found significant by univariate analysis in their study, but were excluded after results of multivariate analysis. These included elevated blood urea nitrogen, adverse American Society of Anesthesiologists (ASA) score, emergency surgery and low serum albumin. Variables that did not reach levels of significance included patient age, gender, anastomotic level, low/high body mass index, diabetes mellitus, history of

smoking, use of drains, intraoperative hypotension, prolonged operating room time, and grade of the operating surgeon.

Alves et al, in their study of large bowel anastomoses found several factors to be associated with the risk of anastomotic leak. These were radiation therapy, ASA score >2, white cell amount > 10,000/$mm^3$, renal failure, recent steroid use, increased operating time, intraoperative septic conditions, abdominal drainage, and perioperative blood transfusion[27].

Pickleman and co workers[30] noted that hypertension was found in a significant number of the patients they analyzed who had undergone small bowel resection with subsequent leak (9 out of 798 patients), although the reason for such an association was not clear to them. They also noticed that cigarette smoking increased leak rates in colonic anastomoses, but not small bowel cases. Analysis of operative blood losses showed no correlation with the occurrence of anastomotic dehiscence.

## Management of Suture Line Disruptions

Anastomotic leakage is associated with a significant mortality rate[17,27] and has also been mentioned as being an independent predictor of mortality[22], despite advancements in the fields of parenteral nutrition and antibiotics.

Patients who have undergone operative intervention on their bowel may have either clinical or sub clinical leaks. Sub clinical leaks are

discovered using routine radiological contrast assessment in the post operative period; the patients exhibiting no other clinical features.

Symptoms of dehiscence may include high grade fever and localized or generalized abdominal pain. Evidences of leakage may be apparent from examination of the patient, which may reveal local or generalized peritonitis, and faecal or purulent discharge from the wound or drain site[12]. Investigations often will reveal leucocytosis, and radiological assessment of the abdomen usually shows the leak (in contrast studies) or its sequelae such as intra abdominal abscess (detectable by sonography or computerized tomography).

Management of patients with anastomotic leak presents a considerable challenge in view of the poor outcomes generally associated with it. Bruce et al[12] suggested that anastomotic leak be divided into 3 categories viz. radiological, clinical minor and clinical major. The former two did not require any change in management or intervention, although the clinical minor group would probably require a greater length of hospital stay. The clinical major group, by definition, would require a change in management, including antibiotics used, delay in resumption of oral intake or prolonged hospital stay. Such a classification, they proposed, would help in clinical audit and epidemiological research.

Patients with intestinal suture line disruption have to be evaluated systematically. Generalized peritonitis warrants relaparotomy, and

reassessment of the anastomotic site. The surgeon usually has to decide between redoing the anastomosis over again, or in more adverse circumstances, creation of a defunctioning stoma to tide the crisis over. The constructions of such a stoma poses more of a problem as the site of the leak occurs more proximally in the bowel, due to the problems associated with the loss of intestinal secretions and subsequent metabolic disturbances.

Localized collections in the abdomen, such as abscesses, can be managed with percutaneous aspiration, usually under radiological guidance. Other essential measures include cessation of oral intake and institution of parenteral nutrition, thus giving the bowel some rest.

In spite of these measures, we find that this condition carries a grave prognosis, and most of the deaths occur in the face of overwhelming sepsis.

## TECHNICAL OPTIONS FOR FASHIONING ANASTOMOSES

Different materials have been used to join the bowel such as catgut and stainless steel for the past 160 years. Newer materials include monofilaments and absorbable sutures. Surgical staplers come into play for the past 30 years. The main drawback is their cost.

## SUTURING: TECHNICAL ISSUES

### Choice of suture material.

Most foreign materials will evoke an inflammatory reaction in the human body. This holds for the suture material, also materials like silk has a potent ability to cause cellular infiltrate as long as 6 weeks. Substances like prolene, catgut evoke a milder response.

Absorbable and non-absorbable sutures have a little difference in maintaining the strength of the anastomosis. The ideal material is the one that elicits little or no inflammatory changes and still maintains the strength of the anastomosis. The newer generation materials like the monofilament sutures and coated braided sutures represent a substantial advantage over the silk and other monofilament materials.

### Continuous versus Interrupted sutures

Both continuous and interrupted techniques have been used for bowel anastomoses. Retrospective reviews have not shown any added

advantage of interrupted sutures over continuous sutures in a single layer anastomosis.

Oxygen tension and blood flow are important factors in anastomotic healing. With continuous sutures the perianastomotic tissue oxygen tension was lower than with interrupted sutures. A prospective trial which compared the continuous single layer with interrupted double layer technique, no significant difference was observed. The added advantages are reduced operating time and cost.

## Single layer versus double layer Anastomoses

A double layer anastomosis done by Travers and Lembert consists of a continuous or interrupted inner layer of absorbable suture and outer layer of interrupted absorbable or non-absorbable suture. A randomized trial comparing the single and double layer techniques showed no evidence of increased risk of leakage in single layer anastomosis. The only added advantage is reduced operating time.

## STAPLING : TECHNICAL ISSUES

### Choice of Staplers

Surgical staplers were first introduced by Hultl in 1908, but they did not gain popularity at that time. But, for the past 30 years, they have changed surgical practice dramatically. With these modern devices failure rate are rare and anastomoses in inaccessible areas are easier to be done.

Three types of staplers are available. The transverse anastomosis (TA) stapler is the simplest. This places two staggered rows of B-Shaped staples across the bowel. They do not cut the bowel and to be divided in a separate step.

The gastrointestinal anastomosis (GIA) stapler which places two double staggered rows of stapler and the bowel is cut between the two rows.

The End to end anastomosis (EEA) staples is a circular stapler with a double rows of staples and cuts the tissue within the staples with a cylindrical knife.

All the staplers are made of titanium which causes little tissue reaction and as they are non-magnetic do not pose a difficulty with MRI scanning.

**Staple Height**

TA and GIA staplers are available with different variety of inserts with different types of staples. These inserts vary in width and height of the closed staple. These staplers are designed for use in particular tissues like inserts designed for closing blood vessels should not be used for bowel and vice versa.

With TA and EEA the depth of the closed staples can be varied by adjusting the distance between the staples and the anvil.

The safe range of closure is indicated by a coloured area on the instrument.

A comparison of anastomotic techniques which used blood flow to divided tissues as outcome showed the best flow to the anastomosis done by stapler in which the staple height was adjusted to the thickness of bowel wall followed by double layer stapled and sutured anastomoses followed by double layer sutured anastomoses and tightly stapled anastomoses.

**Hand Sewn Versus Stapled anastomosis**

Titanium staples provoke only a minimal inflammatory response and provide immediate strength to the edges during the phase of healing.

Tissue eversion of the stapled anastomoses is of important concern as everted hand sewn anastomoses are inferior to inverted ones. However, the greater support and improved blood supply associated with stapling counteract the negative effects of eversion.

In 1993, a randomized trial studied 440 patients who had either hand sewn or stapled anastomosis for ileocolic anastomoses. They were assessed 10 days after operation both clinically and radiologically for presence of leak. The leakage rate in hand sewn group was 8.3% compared to 2.8% in stapled group.

For colorectal anastomoses no significant difference was found in leakage rate, mortality, tumor recurrence rate or wound sepsis.

However, strictures and technical problems are more in stapled group. A possible explanation for higher incidence of leak in the study was due to surgical inexperience and variety of suture techniques in the hand sewn group.

In a study conducted in Scotland, the rate of radiologically proven leakage was higher in sutured group, but no difference was seen with respect to clinical leaks and morbidity / mortality.

Even when anastomoses was done on adverse conditions like cancer, malnutrition etc., no significant differences was detected in the outcome between hand sewn and stapled anastomosis. Cancer recurrence rates at anastomotic site was found to be higher or lower depending on the technique used.

## Unusual Techniques

In 1892, Murphy introduced a button which has two metallic studs used to hold the bowel edges until adhesion occurs. Then the stud is voided through the rectum. Dissolvable polyglycolic acid system are developed. These anastomotic rings leave a gap of 1.5, 2.0 or 2.5 mm between the bowel ends to prevent ischaemia.

Methyl-2-Cyanoacrylate, an adhesive agent used for anastomoses has been studied. There was only moderate inflammation, but the over all leakage rate was high. Fibrin glue in the setting of bowel anastomoses was also studied.

All these substances are not strong enough to hold the bowel. They can be used to coat the sutured bowel anastomoses.

## Bowel Preparation and anastomosis

Mechanical Bowel preparation (MBP) was thought an essential procedure of colorectal surgery for the past 100 years. Various observational studies showed that MBP was associated with reduced morbidity and mortality. The advantages listed are reduction in intraluminal bacterial load, prevention of anastomotic disruption by fecal pellets and easier handing of bowel.

A randomized clinical trial of 2005, studied the effect of MBP on surgery involving left sided colorectal resection with primary anastomosis. These showed an increase in anastomotic leakage in the group that received MBP then the group that doesn't.

Another two trials have been published in 2007, One has 1431 patients who underwent elective colorectal surgery. The leakage rate was 4.87 in the MBP group compared to 5.4% of the non-MBP group which was not significant.

Another group with 1343 patients also found there was no significant differences in the outcome. However, in a meta-analysis which studied 10 randomized trials, the rate of anastomotic leakage and wound infection are found to be higher in MBP group than the non-MBP

group . The most probable explanation is the immune changes in the colonic mucosa that impede wound repair.

Enemas are given to patients planned for anterior resection to ensure that fecal matter does not impede on stapler usage. It is advised to stop eating solid foods 24 hours before surgery.

Many trials confirmed the benefits of using perioperative IV antibiotics. But there is an increased risk of Clostridium difficile diarrhea with usage of cephalosporins, penicillin and clindamycin.

Prophylaxis for thromboembolism is must in all patients undergoing intestinal anastomosis. Mesenteric venous Thrombosis (MVT) accounts for 1/10 of acute mesenteric ischaemic events. The extent of thrombosis may be upto mesenteric infarction requiring urgent repeat laparatomy.

## Controversial issues in Intestinal anastomosis

### *Inversion Versus Eversion*

The technique of inversion described by Lembert versus eversion of the anastomotic line was controversial. The inverting methods ignore the principle of accurately opposing the cut ends. Halsted, who proposed the extramucosal technique shows a low leakage rate. In 1970 trial demonstrated the importance of inverting the cut edges of bowel which showed the rate of anastomotic leaks was higher in the group of everted suture anastomoses than the group with inverted suture anastomoses.

***Nasogastric Decompression***

Routine decompression of stomach in patients undergoing intestinal anastomosis remains controversial. In many randomized controlled trials, the use of nasogastric tube offered no significant advantage in reducing anastomotic leakage. Infact, it increases the risk of respiratory tract in infections. But if the masogastric tube is not used, gastric dilatation can develop which should be addressed.

***Abdominal Drains***

These are two school of thoughts regarding the placement of intra abdominal drains. The first thought in that the placement of intraabdominal drain serves in the diagnosis of anastomotic leakage or bleeding in the early postoperative day. The second thought is that the drains may irritate the peritoneum and increase the production of serous fluid and the drains may provide route of entry for the microbes into the peritoneal cavity. The drain may physically impede the movements of the omentum and thus hinder the body's natural mechanism against any infection. Drains also have a high degree of blockage.

One study showed the increase in anastomotic leakage after the placement of drains in dogs. In 1999 another study studied the placement of pelvic drains in patients undergoing rectal or anal anastomosis which showed no improvement in outcome.

Inspite of all these, many surgeons today practice the placement of drains for rectal or anal anastomosis, since the risk of fluid collection is higher. In emergency operation, where anastomosis is done drains may be indicated where peritonitis is present.

# OPERATIVE TECHNIQUES

## Patient positioning and Incision

Most of the abdominal procedures are done with the patient in supine position and performed with an adequate midline incision. For procedures in the pelvis, the patient is put in lithotomy position with adequate access to the anus. Care must be taken not to excessively flex or abduct and adequate padding to be given to prevent thrombosis and neuropraxia. For procedures of oesophagus, the patient is usually positioned on one side as the incision of choice is lateral thoracotomy.

The table can be tilted during the course of surgery. A $30^{o}$ head down (Trendelenburg position) may be helpful for pelvic procedures as it moves the moving small bowel out of the field.

## Exposure, Mobilization and Dissection

The incision for abdominal operative procedure should be made in such a way to allow adequate access ,with the use of retractors lateral aspect of field can be exposed. Operating in pelvis is usually difficult for the surgeon. The small bowel is usually packed with the help of wet gauge to get clear view of the pelvis. If the bowel is free like the small bowel, transverse colon, sigmoid colon, it is easily brought to the surface and rest all the contents can stay inside the peritoneal cavity. With other

sections of the bowel like ascending. descending colon the lateral peritoneal borders should be divided to bring the structures to the surface. Tension of the anastomoses is the problem in case of oesophagus and colon and care to be taken that they lie together easily.

**Bowel resection**

The segment of the bowel to be resected is cleared off from surrounding adhesions. First the mesentery is divided by preserving the blood supply to the ends of the bowel wall. This is very easy in case of small bowel than large bowel because of the ample blood supply to small bowel. The surrounding fat and appendices epiploicae are removed in case of large bowel.

Achieving hemostasis is very important which is usually done by tying knots. Sometimes the knots placed close to the bowel can cause distortion or angulation of the bowel, thereby disturbing the anastomosis. They may slip sometimes, which may result in hematoma within the leaves of the mesentery which may hinder the viability of the bowel. It is safe to use a fine stitch to secure anastomosis. A ultrasonic scalpel or bipolar electrocautery can be used.

**Division of bowel**

The bowel segments to be resected is isolated with the use of non crushing clamps placed some distance from the diseased segment. Crushing clamps are then used on the diseased side of the bowel. The

bowel is then divided in between the crushing and non-crushing clamps with the help of a knife. This prevents the contents of the diseased segment from leaking into the peritoneal cavity.

Using staplers for division of the bowel, has an added advantage as it seals the cut edges.

**Single Layer Sutured ExtraMucosal –**

**Side to side Enteroenterostomy**

Side to side anastomosis is done as a bypass procedure like gastroenterostomy, when there is a discrepancy in the diameter of the resected edges as in case of ileocolic anastomosis done after right hemicolectomy.

Two stay sutures with 3–0 polyglycolic acid are placed on the antimesenteric border 8cm apart. An enterostomy of length 5cm is made on each loop. This is made either with the use of blade or electrocautery.

Then a full length sero-muscular and submucosal stitch with 4-0 polyglycolic acid is placed 5 to 10mm from the enterotomies. The stitch avoids the mucosa as this helps in epithelization. An over and over stitch is started and taken in the direction of the surgeon with proper inversion of the suture line. At the proximal ends of the anastomosis baseball stitch is made around the anterior wall of the anastomosis. Connell stitch is used to invert the anterior layer.

Another 4–0 polyglycolic acid full thickness seromuscular and submucosal suture is taken in the same location. The remaining posterior wall is sewn in the same manner and corners approximated with baseball stitch. Then the anterior layer is sewn in the same manner with the Connell stitch or an over and over stitch.

**Double layer end to side Enteroenterostomy/Enterocolostomy**

This is done in case of ileotransverse anastomosis. The proximal small bowel is brought in apposition with the side of the colon not far than 2.5 cm to 5 cm from the blind end. This is to prevent the blind loop syndrome.

Stay sutures of 3–0 polyglycolic acid are taken from the proximal limb 10 to 15mm from the cut and the distal limb. Interrupted seromuscular sutures are taken between the proximal and distal limb. About three to six for a centimeter spacing is taken and tied sequentially.

An enterotomy or colotomy is created on the distal limb, then a full thickness 3–0 polyglycolic acid suture is taken in the posterior wall and sewn in an over and over stitch toward the surgeon. A second full thickness suture is taken at the same point and sewn away from the surgeon to complete the anterior layer. The corners are rounded with baseball stitch and anterior layer with Connell stitch.

Another series of interrupted seromucular stitches are taken anteriorly just like that was taken posteriorly. Care must be taken not be

narrow the lumen of anastomosis. The lumen is palpated for confirmation of patency.

If there is any mesenteric defect, it should be closed.

## Double Layer End to End anastomoses

This begins with placement of interrupted submucosal sutures on the posterior aspect of the anastomosis. The suture are tied when this layer is complete.

For inner layer of sutures usually with an absorbable material begins in the antimesenteric end. The sutures are taken seromuscular and submucosal. A continuous over and over technique is followed for the posterior aspect of the anastomosis. The mesenteric corner of anastomosis is secured by Connell technique. The anterior aspect is either sewn by over and over stitch or Connell technique can be used.

Another layer of interrupted, nonabsorbable sutures on the anterior outer aspect and the anastomosis is completed.

## End to End Ileocolic anastomosis

This is achieved by widening the orifice of the smaller lumen. The outer layer of submucosal sutures is inserted in an oblique fashion away from the cut edge of the bowel on the antimesenteric aspect in the end of smaller calibre.

# MATERIALS AND METHODS

This prospective, analytical study was conducted in the Department of General Surgery, Madurai Medical College between November 2011 – October 2012. All patients above the age of 12 years undergoing surgical closure of a full thickness small intestinal breach, who satisfied the inclusion criteria, were analyzed and followed up until their discharge from hospital or death.

These patients were ultimately divided into two groups :

- Group 1 – (cases): Patients undergoing small bowel surgery with subsequent suture line disruption.
- Group 2 – (controls) : Patients undergoing small bowel surgery without subsequent suture line disruption.

## Our exclusion criteria included

1. Duodenal surgery
2. Anastomoses between the small intestine and stomach or bilary tract.
3. Feeding jejunostomies.
4. Death of the patient within 28 days of admission due to causes other than suture line disruption.

**Detection of suture line disruption was based on the following :**

- Demonstration by relaparotomy.
- Demonstration by dye or contrast studies.
- Efflux of bowel contents from the wound or drain.
- Demonstration of any localized collection of bowel contents in the abdominal cavity by ultrasonography guided aspiration.

## Methodology

The patients in the study were initially interviewed with specific regard to certain factors known to be of importance in the etiology of disruption. These included age, sex and duration of symptoms before presentation to hospital (in emergency cases). The use of tobacco, alcohol and steroid medications if any was noted, and the presence of Chronic Obstructive Pulmonary Disease (COPD), Diabetes Mellitus, and systemic hypertension was looked for. The attending surgeon's pre operative diagnosis was also noted down, as per the patient records.

The vital parameters of each patient viz., pulse rate, blood pressure and respiratory rate were recorded at admission. Anthropometric assessment was subsequently done. The patient's weight, height, mid arm circumference and triceps skin fold thickness were recorded.

Laboratory data obtained in each patient included hemoglobin, serum total protein, albumin, urea, creatinine and electrolytes. Blood was sent for culture and enteric fever serology (Widal) where indicated.

Intra operative variables studied included the presence of gross peritoneal contamination, the nature of the peritoneal contaminant, the site of pathology in the small bowel, the presence, number and size of perforations, the vascular supply at the region of the surgical closure, the luminal disparity between anastomosed ends of bowel, the suture material used, presence of any distal obstruction, and the ease of surgical closure. The amount and nature of intraoperative fluids given the presence of any adverse intraoperative hemodynamic event, the use of drains, the grade of the operating surgeon and the intraoperative diagnosis were also noted.

Post operatively, several factors were taken into account such as use of vasopressor support, ventilatory support, antibiotics used, use of steroids, presence of wound infection, and the time of starting oral fluids.

All data was recorded using a structured proforma (Appendix – 2).

Patients who had documented suture line disruption and underwent relaparotomy for the same were taken as fresh cases, and data recorded again as above.

The end point of the study was with the discharge of the patient from hospital, or at the time of the patient's death, as the case may be.

Patients who had suture line disruption (test) were compared with patients whose anastomoses did not leak (control). Statistical analysis was done using Fisher's exact test and chi square test for discrete variables and the student's t-test for continuous data. The Statistical Package for Social Sciences (SPSS) software version 10.0 was utilised. Factors found to be having a significant association with the occurrence of suture line disruption were considered for further analysis by binary logistic regression, so as to identify independent risk factors for the outcome. The Odd's ratio was calculated for each of these variables. A p value $<0.05$ was considered significant for the purpose of this study.

# OBSERVATIONS

This study was conducted over a period of 12 months, from November 2011 to October 2012. A total of 73 patients (with 83 anastomoses) were included in the study, the majority of them being emergencies (96.3%). As per our inclusion criteria, all entero-enteric and entero-colic anastomoses were analyzed for factors that could predispose to suture line disruption.

Thirty two anastomotic disruptions were subsequently noted in 25 subjects. Forty eight patients with 51 anastomoses, who made an uneventful postoperative recovery, formed the control group. All anastomoses were hand sewn and constructed in 2 layers.

## Patient Variables

### *Age distribution (Fig4) and Gender distribution (Fig.5)*

The age of subjects in this study ranged from 13 to 70 years, with a mean of 38.02 ± 16.26 years. The mean age of patients in the leak group was 39.63 ± 16.73 years whereas the corresponding value for non leak group was 37.02 ± 16.03 years. This difference between groups was not significant (p=0.481).

Seventeen men (37.7% of all males) and 8 women (28.6% of females) had anastomotic disruption. There was no significant difference between men and women for the occurrence of leak (p=0.363).

***Duration of symptoms (Fig.6)***

The duration of symptoms ranged from 6 to 288 hours, with an overall mean of 89.15 ± 67.23 hours. The mean duration of symptoms in the disruption group was 106.85 ± 81.21 hours as compared to 78.43 ± 56.93 hours in the control group, this difference was not significant (p=0.081).

***Co Morbid factors***

In the case group, 1 patient had Diabetes Mellitus, 1 had systemic hypertension and 1 had Chronic Obstructive Pulmonary Disease, as compared to the control group where the corresponding values were 2, 1 and 3 respectively.

8 of 25 patients in the leak group and 17 of 48 in the control group were smokers. This difference was not significant (p=0.770). Similarly, history of alcohol intake was noted in 7 of the leak group and 10 of the control group (p=0.492).

***Admission parameters***

The mean pulse rate per minute at presentation between cases and controls did not differ significantly (98.99 ± 12.09 Vs. 95.02 ± 17.54 respectively; p=0.279). Neither mean systolic blood pressures (mm Hg)

nor mean respiratory rates (per minute) amongst case and control groups showed any significant differences (109.50 ± 18.29 Vs. 110.90 ± 19.36 and 20.50 ± 6.98 Vs. 18.78 ± 5.89 respectively; p=0.741 and 0.233 respectively).

American Society of Anesthesiologists' (ASA) grade of 3 or more was significantly associated with the occurrence of anastomotic disruption (p=0.049) (Table 1).

***Anthropometric parameters***

The mean mid arm circumference (MAC) amongst cases (24.91 ± 5.71 cms) was not significantly different from the mean values in the control group (23.57 ± 3.59 cms). Similarly, the mean body mass index and triceps skin fold thickness between groups did not differ significantly (21.27 ± 3.22 $Kg/m^2$ and 7.35 ± 1.45mm in cases, Vs. 20.77 ± 3.04 $Kg/m^2$ and 7.37 ± 0.98mm in controls, respectively).

***Hematological and biochemical parameters (Table 2)***

Hemoglobin values between leak and control groups showed a significant difference (p=0.031), the mean values for the former being 9.509 ± 1.941 gm %, and 10.478 ± 1.967 gm% for the latter.

Serum albumin was significantly lower in the leak group (2.772 ± 0.450 gm %) as compared to the control group (3.149 ± 0.609 gm%) (p=0.003).

Pre operative serum sodium levels were found to be significantly lower in the leak group (131.09 ± 6.32 meq/L) compared to control values (135.63 ± 7.51 meq/L) (p=0.008). The mean serum total protein, urea and creatinine levels were comparable in both the groups.

## Indications for small bowel surgery (Table 3)

Of the 83 cases in our study, small bowel perforation was found in 28 (33.7%) individuals, small bowel gangrene in 16 (19.3%) and intestinal obstruction in 33 (39.8%). Patients undergoing reanastomosis for suture line disruption comprised 6 (7.2%) cases. No single group has a significant association with the occurrence of leak except the reanastomosis group (p=0.030).

The 28 small bowel perforations in our study included 25 spontaneous and 3 traumatic perforations. Of the 33 cases of intestinal obstruction, 21 were adhesive in nature, 3 were obstructed hernias, and 4 were due to intra abdominal tuberculosis, 2 due to right sided colonic malignancy and 3 due to Meckel's diverticulum.

Of the 22 perforations in which the Widal test was done, 7 were positive. This group was not associated with leakage more than their Widal negative counterparts (p=1.000) (Table 4).

## Intra operative Variables (Table 5)

The duration of surgery in the study ranged from 60 to 300 minutes, with a mean of 143.31 ± 57.01 minutes. The mean duration of surgery in both the groups differed significantly (158.91 ± 62.70 minutes) in the leak group Vs. 133.53 ± 51.37 minutes in the controls (p=0.048).

The presence of gross peritoneal contamination, as evidenced by the finding of food, bile, pus or fecal matter in the peritoneal cavity, was significantly associated with the occurrence of leak (p=0.023).

The nature of the peritoneal contaminant did not have a significant bearing on the healing of the suture line (p=0.177). The impact of fecal contamination alone on the incidence of leak with respect to the others (pus, bile and blood) was also not significant (p=0.50).

The number of perforations had no association with suture line disruption (p=0.326). The mean distance at which all failed distal small bowel anastomoses were constructed from the ileo-caecal junction was 37.35 ± 25.86 cms, as compared to the value of 45.49 ± 33.38cms in the control group. This difference was not significant (p=0.322). Similarly, the mean distance from the duodenojejunal flexure at which unsuccessful

proximal small bowel anastomoses were constructed was 37 ± 26.6 cms, while successful cases had a mean of 38.33 ± 33.04cms. This difference also did not attain significance (p=0.936).

All the suture lines were constructed in two layers in the study. Non absorbable sutures were used for both layers in 16 cases, whereas full thickness absorbable with seromuscular non absorbable was used in 67 cases. Leaks were observed in 6 and 26 cases respectively in these groups. This difference was not statistically significant (p=1.000) (Fig. 7). The absorbable material used was Polyglycolic acid and non absorbable used was silk in all the cases.

A total of 49 ileoileal, 13 ileocolic, 19 jejunojejunal and 2 jejunocolic anastomoses were constructed in the study period (Fig. 8). End to end anastomoses were performed in 66 patients, end to side in 10 cases and wedge resection anastomoses in 7 cases, with a leak frequency of 23, 7 and 2 respectively. The level of the anastomoses (i.e. enteroenteric or enterocolic) did not have a significant association with the occurrence of suture line disruption (Table 7). Seven of the 10 end to side enterocolic anastomoses that were constructed leaked, whereas 2 of the 5 end to end enterocolic anastomoses leaked (p=0.329).

The mean amount of fluids infused intra operatively in the leak group was 2908.44 ± 1272.50ml and 2368.63 ± 1024.84ml in the control group. This difference was significant (p=0.037). However, the mean amount of crystalloids, colloids and blood individually infused between groups was comparable (p values 0.151, 0.452 and 0.259 respectively). Intra operative hypotension (taken as a drop of the systolic blood pressure to 80mm Hg or less) occurred in 9 patients, out of whom 8 had anastomotic dehiscence. This was statistically significant (p=0.001).

The usage of drains was found to have an association with the occurrence of suture line dehiscence in patients undergoing primary anastomoses (p=0.029).

The majority of surgeons who operated upon the patients in this study were those having more than 3 years of surgical experience. Neither the anastomoses constructed by this group nor those done by surgical trainees (having less than 3 years experience) were significantly associated with suture line disruption (p=1.000).

## Post Operative Variables (Table 7)

### *Ventilatory and vasopressor support*

The use of ventilator support in the immediate postoperative period had a significant association with occurrence of leak (p=0.019) whereas the use of vasopressor support had no such association (p=0.118).

### *Time of starting oral intake*

The mean time before resumption of oral feeds was 127.89 ± 41.19 hours in the leak group, whereas in the control group it was 115.1 ± 28.97 hours. This difference was not statistically significant (p=0.153).

## Logistic Regression Analysis (Table 8)

Risk factors found to be of significance by univariate analysis were subjected to binary logistic regression in order to find independent risk factors for the occurrence of suture line disruption. In this manner, 4 factors viz., low serum albumin, low preoperative serum sodium, gross peritoneal contamination and Intraoperative hypotension were found to be significantly contributing to the risk of anastomotic leakage.

## Releak and Mortality Data

All deaths (13) occurred in the group of 25 patients who had suture line disruption. In this leak group, 11 (44%) were managed conservatively and 4 patients subsequently died. Fourteen patients (56%)

were reoperated. Four (16%) had an exteriorization of the leak site done, and 1 patient in this group died. The remaining 10 patients (40%) underwent freshening of the bowel edges at the site of the primary anastomoses followed by reanastomosis, of which 8 patients ultimately died, 4 of these patients died in the early post operative period. Of the remaining 6 patients, 5 had demonstrable releak and 4 ultimately died. In the group of patients who died, no significant different was observed in terms of mortality between those who were managed conservatively and those who had an exteriorization of the leak site (p= 1.00). However, those who underwent reanastomosis for anastomotic leakage in this group had a significant association with mortality (p=0.041) (Fig. 9).

Factors found to have a significant association with mortality by univariate analysis included low serum albumin (2.71 ± 0.48 Vs. 3.11 ± 0.57 g%; p =0.022), increasing duration of surgery (165 ± 56.12 Vs. 130.5 ± 45.15 minutes; p=0.02), ASA grade of 3 or above (p=0.005), intraoperative hypotension (p=0.004), ventilatory support (p=0.13), reanastomosis for leak (p=0.008) and small to large bowel anastomoses (p=0.005) (Table 9).

***Table 1 : ASA grade and leak***

| ASA grade | Leak | | Total |
|---|---|---|---|
| | Present | Absent | |
| ASA grade equal to or more than 3 | 19 | 19 | **38** |
| ASA grade less than 3 | 13 | 32 | **45** |
| **Total** | **32** | **51** | **83** |

P=0.049

***Table 2 : Biochemical Parameters and leak***

| Variables | Leak | | p value |
|---|---|---|---|
| | Present | Absent | |
| Hemoglobin (g/dL) | 9.509 ± 1.941 | 10.478 ± 1.967 | 0.031* |
| Serum total protein (g/dL) | 5.391 ± 1.242 | 5.808 ± 1.094 | 0.112 |
| Serum albumin (g/dL) | 2.772 ± 0.450 | 3.149 ± 0.609 | 0.003* |
| Urea (mg/dL) | 50.34 ± 39.00 | 47.53 ± 34.58 | 0.732 |
| Creatinine (mg/dL) | 0.86 ± 0.386 | 0.805 ± 0.630 | 0.991 |
| Sodium (meq/L) | 131.09 ± 6.32 | 135.63 ± 7.51 | 0.006* |
| Potassium (meq/L) | 3.912 ± 0.653 | 4.105 ± 0.530 | 0.146 |

***Table 3 : Indications for surgery and leak***

| Post-op diagnosis | Leak | | Total |
|---|---|---|---|
| | Present | Absent | |
| Perforation | 13 | 15 | **28** |
| Gangrene gut | 5 | 11 | **16** |
| Intestinal obstruction | 9 | 24 | **33** |
| Reanastomosis for leak | 5 | 1 | **6** |
| **Total** | **32** | **51** | **83** |

P= 0.047

***Table 4 : Typhoid perforations (Widal positive) and leak***

| Enteric Perforations | Leak | | Total |
|---|---|---|---|
| | Present | Absent | |
| Typhoid | 3 | 4 | **7** |
| Non typhoid | 8 | 7 | **15** |
| **Total** | **11** | **11** | **22** |

P= 1.000

***Table 5 : Intraoperative factors and leak rate***

| Variables | Leak | | p value |
|---|---|---|---|
| | Present (n = 32) | Absent (n = 51) | |
| Mean duration of surgery (mins) | 158.91 ± 62.70 | 133.53 ± 51.37 | 0.048* |
| Presence of gross peritoneal contamination | 24/32 | 25/51 | 0.023# |
| Patients with multiple perforations (n = 12) | 7/12 | 5/12 | 0.731 |
| Mean distance from ileocaecal junction in distal small bowel anastomoses (cms) | 37.35 ± 26.86 | 45.49 ± 33.38 | 0.322 |
| Mean distance from duodenojejunal flexure in proximal small bowel anastomoses (cms) | 37 ± 26.6 | 38.33 ± 33.04 | 0.936 |
| Suture material used (inner absorbable and outer non absorbable) | 26/32 | 41/51 | 0.9 |
| Total fluid infused (ml) | 2908.44±1272.50 | 2368.63±1024.84 | 0.037 |
| Presence of Intraoperative hypotension | 8/32 | 1/51 | 0.001 |
| Use of drains (primary anastomoses group) | 21/24 | 30/49 | 0.029 |
| Cases operated by junior residents | 10/32 | 17/51 | 1.000 |

# Chi square test
* Student's test

***Table 6 : Postoperative factors and leak***

| Variables | Leak | | p value |
|---|---|---|---|
| | Present | Absent | |
| Ventilatory support | 10/32 | 5/51 | 0.019* |
| Vasopressor support | 8/32 | 5/51 | 0.118 |
| Time of starting oral intake (hours) | 127.89 ± 41.19 | 115.1 ± 28.97 | 0.153 |

***Table 7 : Anastomotic procedure and leak***

| Anastomosis | Leak | | Total |
|---|---|---|---|
| | Present | Absent | |
| Small to small bowel | 23 | 45 | **68** |
| Small to large bowel | 9 | 6 | **15** |
| **Total** | **32** | **51** | **83** |

***Table 8 : Parameters associated with mortality***

| Variable | Dead (n = 13) | Alive (n = 60) | p-value |
|---|---|---|---|
| Serum albumin (gm%) | 2.715 ± 0.486 | 3.117 ± 0.576 | 0.022* |
| Duration of surgery (minutes) | 165.00 ± 56.12 | 130.50 ± 45.15 | 0.02* |
| ASA grade equal or above 3 | 11/13 | 24/60 | 0.00# |
| Intraoperative hypotension | 5/13 | 3/60 | 0.004# |
| Ventilatory support | 5/13 | 5/60 | 0.013# |
| Reanastomosis for leak | 4/1 | 2/60 | 0.008# |
| Small to large bowel anastomoses | 6/13 | 6/60 | 0.005# |

* student's test # chi square test

***Table 9 : Multiple logistic regression for suture line disruption***

| Variable | B | Standard error | p value | Exp(B) or Odd's ratio |
|---|---|---|---|---|
| Albumin | 1.587 | 0.554 | 0.004* | 4.901 |
| Sodium | 0.134 | 0.047 | 0.004* | 1.144 |
| Peritoneal contamination | 1.422 | 0.636 | 0.025* | 4.147 |
| Intraoperative hypotension | 2.3 | 1.141 | 0.044* | 9.977 |
| Constant | 20.958 | 6.828 | 0.002 | |

Z = Probability of suture line disruption

= 20.958 + (1.587) Albumin + (0.134) Sodium + (1.422) Peritonitis +

(2.3) Introperative hypotension)

***Figure-4 : Age distribution (p=0.338)***

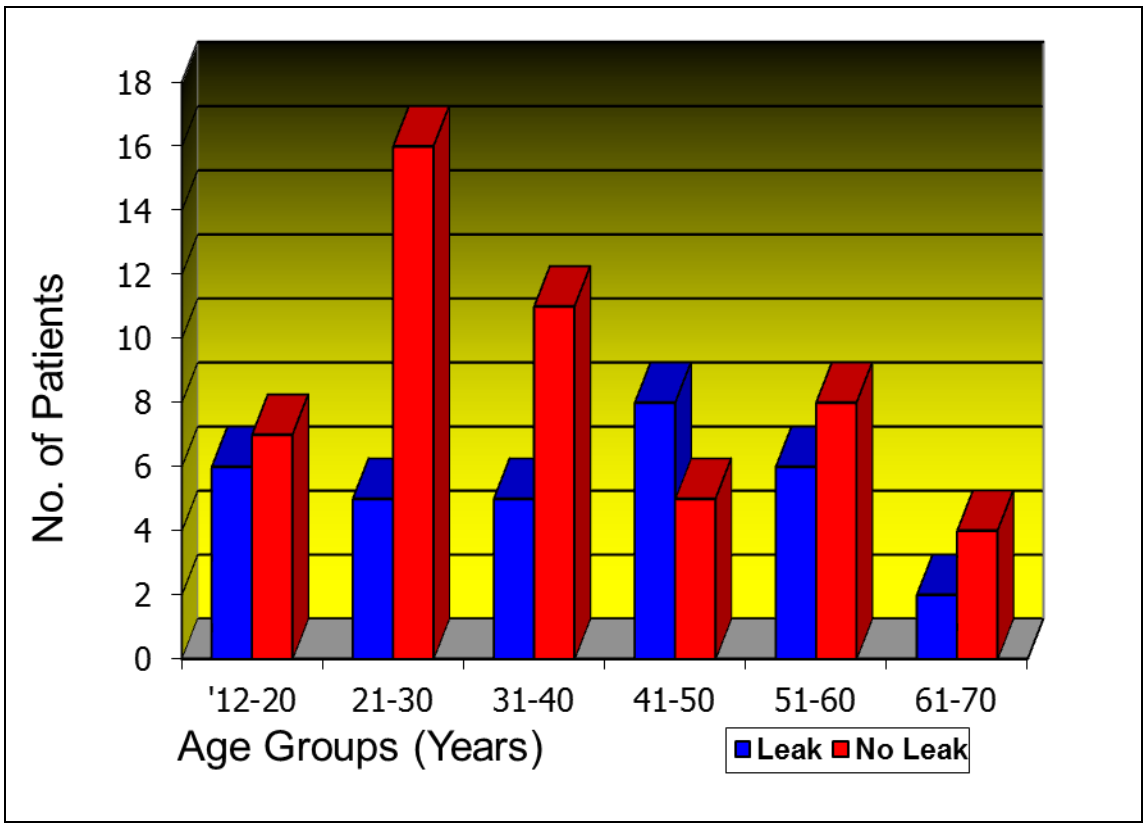


***Figure-5 : Gender distribution***

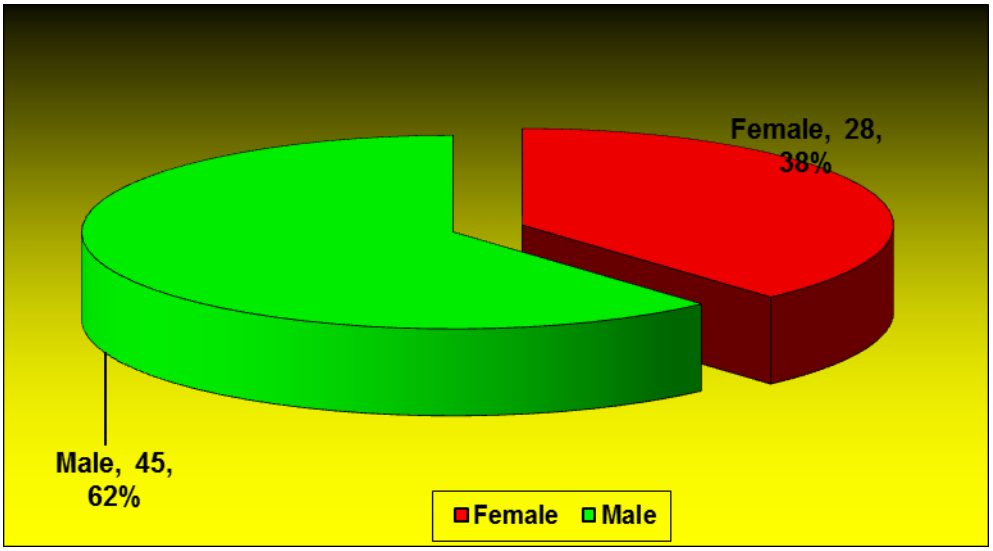



***Figure-4 : Duration of symptoms (p=0.292)***

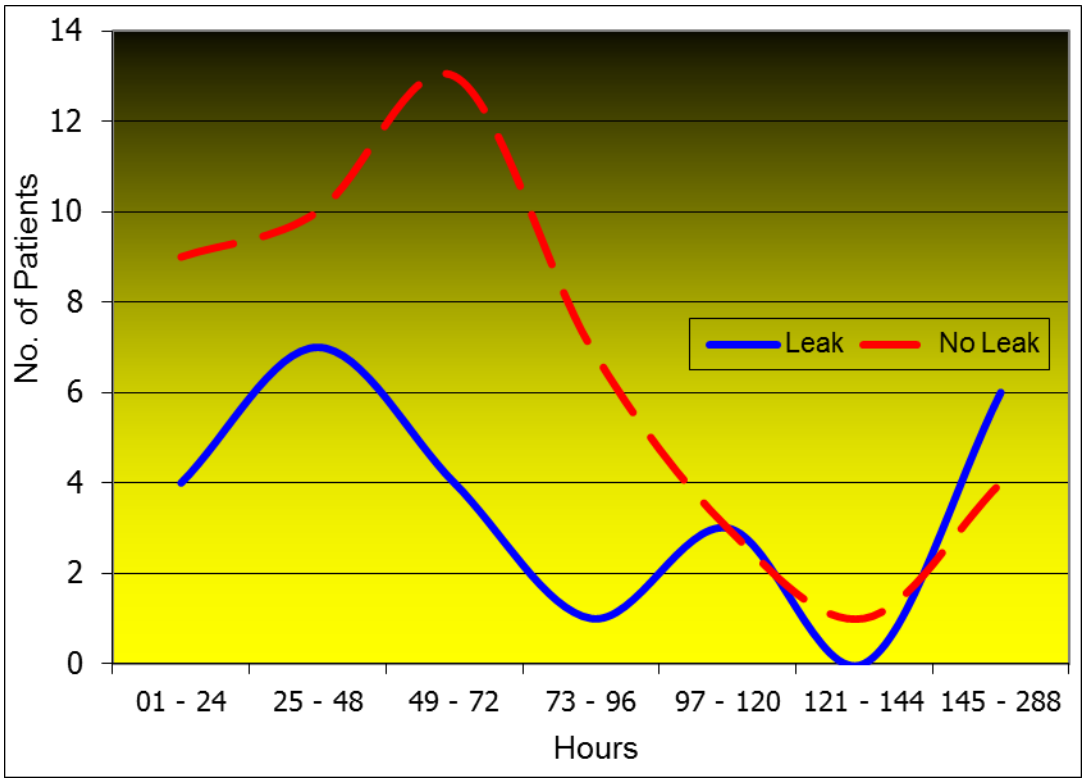
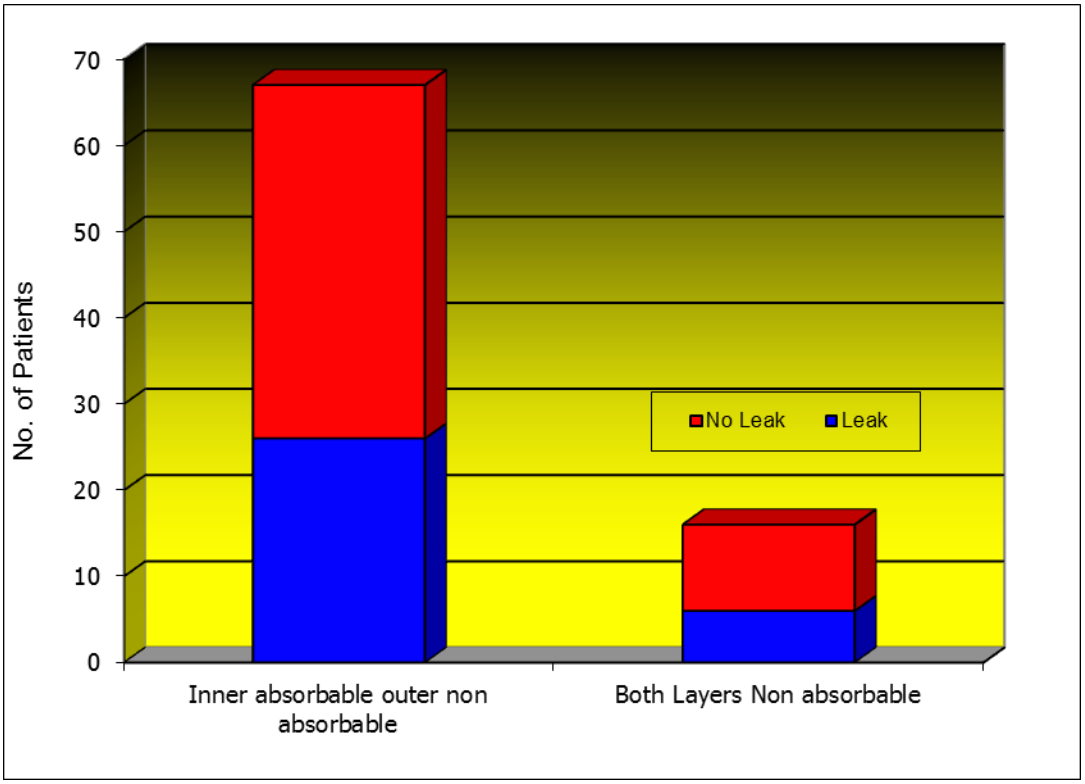


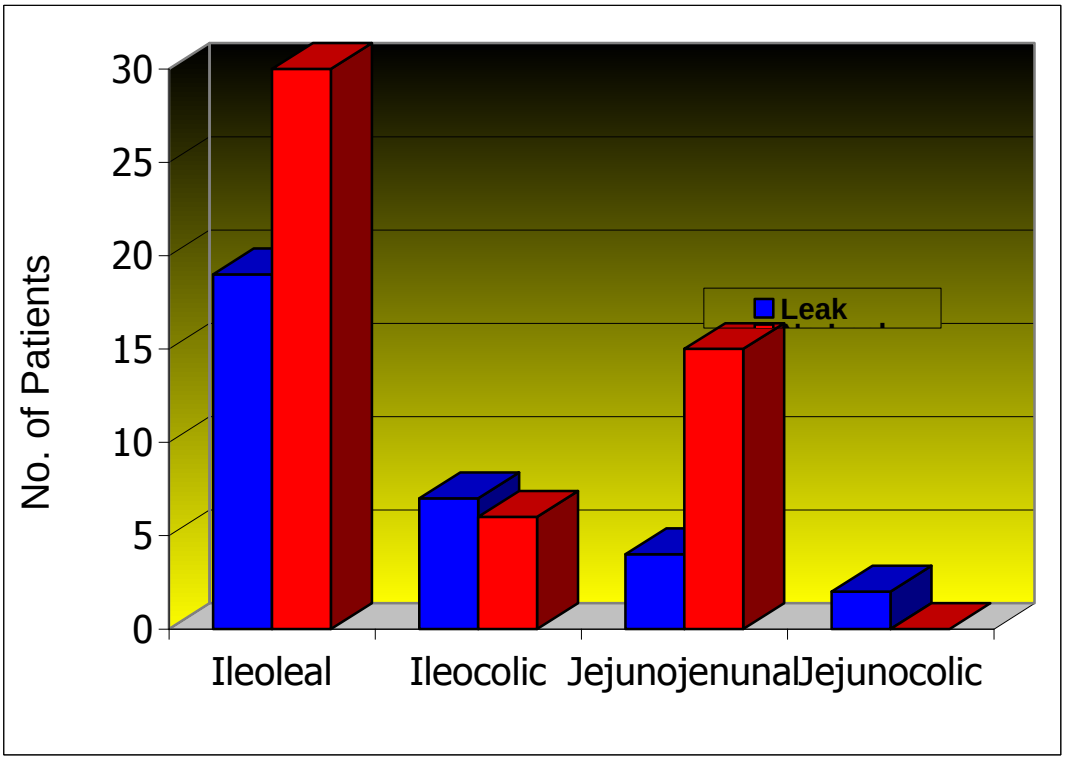
***Figure-7: Suture material and leak (p=0.923)***




## Suture Material

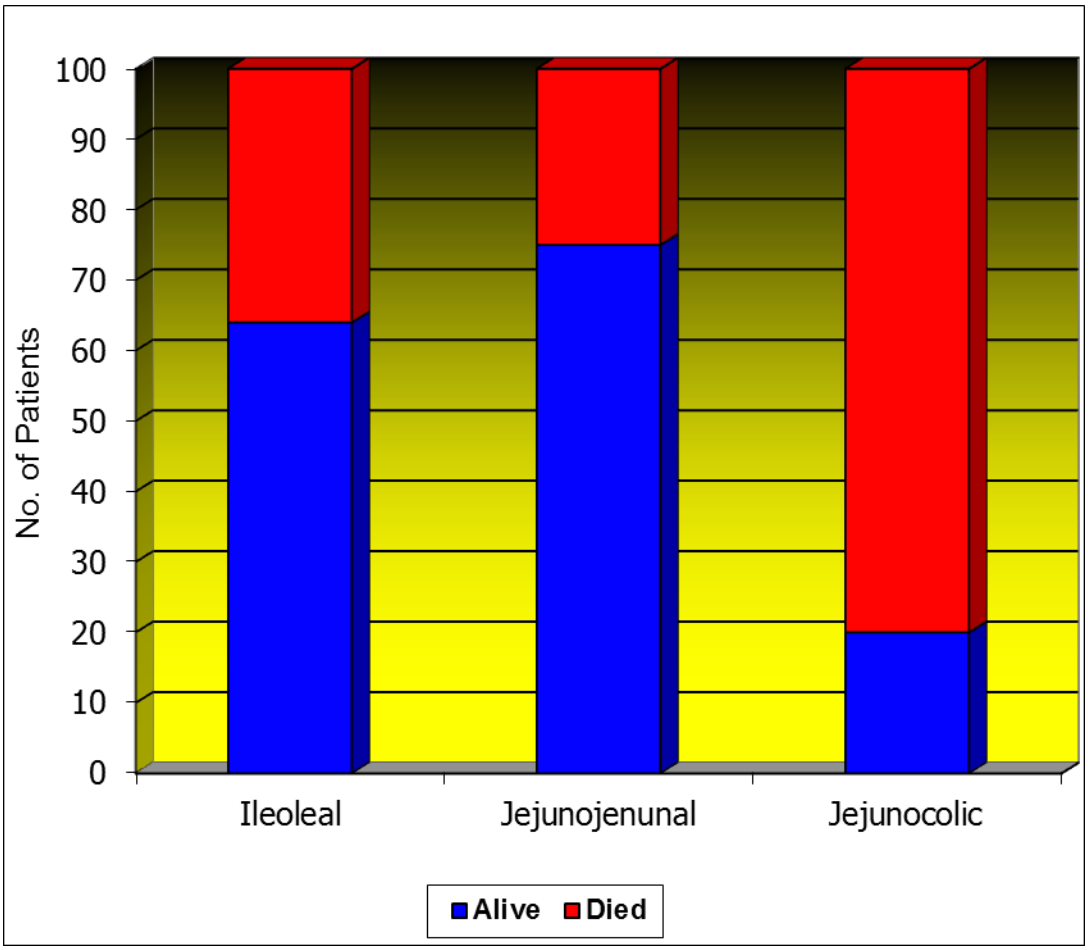
| | Both Layers Non absorbable | Inner absorbable outer non absorbable |
|---|---|---|
| No Leak | 41 | 10 |
| Leak | 26 | 6 |

***Figure-8 :Anastomotic procedure and leak (p=0.074)***




| | ileoleal | ileocolic | jejunojenunal | jejunocolic |
|---|---|---|---|---|
| No Leak | 30 | 6 | 15 | 0 |
| Leak | 19 | 7 | 4 | 2 |

***Figure- 9 : Management of suture line disruption (*p=0.041)***




| | ileoleal | jejunojenunal | jejunocolic* |
|---|---|---|---|
| Died | 4 | 1 | 8 |
| Alive | 7 | 3 | 2 |

# DISCUSSION

The construction of an anastomoses is fraught with potential problems for the surgeon, who realizes that anything short of meticulous attention to it can have devastating consequences. However it is seen that anastomotic disruptions occur frequently despite the greatest attentions to technical detail. This has stimulated many workers to search for other causative factors that although not readily apparent, may be amenable for correction in the perioperative period, thus lessening the incidence of the problem.

This prospective study spanning 12 months had 73 patients with 83 anastomoses between them. Most of the existing work on this subject has been in the form of retrospective studies and have focused on multiple levels of the gastrointestinal tract, rather than a specific part.[13, 17, 22, 30]. To our knowledge, there has been only one study till date[33] which has specifically addressed small bowel anastomotic leakage, but this pertained to its incidence and treatment. No studies have so far dealt with the risk factors per se involved in small bowel suture line disruption.

Our present effort was conducted to clarify issues relating to factors causing small bowel anastomotic leakage with specific reference to emergency surgeries, as the majority of cases (96.3%) we analyzed were emergencies. We utilized logistic regression and multivariate analysis to identify independent predictors of anastomotic leak. This

methodology has been used only in a handful of studies till date, covering both small and large bowel anastomosis[22], or large bowel anastomoses alone[27].

## Age and Gender distribution

The mean age of the subjects in our series was 38.02 ± 16.26 years. Other studies[22] have reported higher mean ages, but none have conclusively stated that advanced age is a risk factor for suture line leak. Hesp and co workers[33] subdivided the small bowel anastomoses into 4 groups based on etiology. Those patients forming the 'inflammatory' group of intra abdominal infections and inflammatory bowel disease had a mean age of 41 years, which compares well with our series. 'Vascular' cases comprised mainly by strangulated hernias and mesenteric vascular occlusions had a higher mean age of 53 years. Stoop and co workers[18] found from their animal studies that advanced age is not an independent risk factor for the breakdown of intestinal anastomoses. The lower mean ages we encountered could be due to the higher prevalence of specific and non specific small bowel perforations in our series, which is usually seen to affect individuals in the prime of their lives. This sub group formed 33.7% of our cases, and the mean age observed was 33.07 ± 14.32 years. Chaikof[41] noted a mean age of 51.8 ± 21.8 years in his work on non traumatic small bowel perforations, which pertained mainly to non infective causes. Chatterjee et al[42] found that non typhoid enteric

perforations in their study occurred mainly in the second and third decades of life, an observation similar to that noted in their work on typhoid ileal perforations[37]. Other studies[43,44] on typhoid enteric perforations have also yielded similar results. Higher mean ages have been reported in other studies on large bowel anastomosis,[17,27] but this could be a reflection of the higher incidence of colorectal malignancies in those series.

Forty five males (61.6%) and 28 female patients (38.4%) constituted our study group. No significant association with leakage was noted in either group. Golub et al[22] made similar observations, noting no gender association for leakage.

## Duration of symptoms

Since our series focused mainly on emergency cases, we felt the need to highlight the possible importance of duration of preoperative symptoms in association with leakage. However, our data failed to reveal any significance between both groups in this regard (p=0.081). A scrutiny of previous studies[13,17,27,30] did not reveal any observations in this regard, although one does mention that the number of preoperative hospital days was not significantly associated with disruption[22].

## Co Morbid factors

The presence of Diabetes Mellitus, Chronic Obstructive Pulmonary Disease (COPD) or Systemic Hypertension were found to have no

association with the occurrence of leaks in our sample population, though the numbers were small. Similarly results were obtained when analyzing history of smoking and alcohol intake in our patients.

Fawcett and colleagues[38] noted that smoking and hypertension were significantly associated with the occurrence of suture line disruption in colonic anastomoses. This was attributed to the increased incidence of microvascular disease in the anastomotic region caused by these risk factors. It is unclear to us whether such factors affect small bowel vascularity too; our sample size did not allow us to make any relevant conclusions in this regard. Pickleman et al[30] also found that hypertension was a risk factor in the development of small intestinal anastomotic leakage. Diabetes has been shown in many studies not to have a significant association with disruption[13,22,30,38] COPD was noted to be an independent predictor of leakage in one study[22].

## Patient parameters

No significant association between baseline hemodynamic measurements at admission in emergency cases and the occurrence of leak or death was noted in our study. As most of the existing literature focuses on a rather uniformly mixed patient population of elective and emergency cases, related observations were lacking in them.

The implications of malnutrition on anastomotic healing have been well established in previous studies. We found that anthropometric

variables possessed no reliability in predicting leaks. That the former is not of use in the assessment of protein malnutrition was already shown by Collins and associates[24] in their study.

A low serum albumin was noted to be predictive of anastomotic disruption in our series (p=0.003). Our findings corroborate the conclusions of other workers who noted the association between hypoalbuminemia and deranged wound healing[28, 29]. Irvin[17], Golub[22] and their colleagues also noted the deleterious effects on low serum albumin levels on anastomotic healing.

The pre operative serum sodium was also shown in our study to have an association with anastomotic leakage (p=0.006). The mean sodium level in the leak group was significantly lower than in controls. Our explanation for this finding is the possible occurrence of anastomotic oedema in the hyponatremic state, which is known to have adverse effects on intestinal suture lines. Chan and co workers[45] concluded from their experimental work that excess water and sodium infused intraoperatively were sequestered in the gut after small bowel surgery, leading to tissue oedema and poor anastomotic healing. We feel that in the setting of low serum sodium levels, water logging of an anastomosis is a possibility.

The detrimental effects of acute uremia on anastomotic healing were exhibited by Colin et al[39] in their animal study. They noted that bursting pressures of midline abdominal incisions and small intestinal

anastomoses were reduced by the uremic state. The degree of fibroblastic growth and cellular proliferation was severely affected by high serum urea levels. None of our patients had established acute renal failure, although pre renal azotemia was noted in some. The mean urea levels between the leak and control groups in our study were comparable.

An ASA grade of 3 or more was associated with increased risk of leakage (p=0.049). Golu[22], Alves[27] and co workers reported the association of increasing ASA grade with anastomotic complications.

## Aetiological factors

The patients in our study were grouped into 4 main categories based on intraoperative findings viz., small bowel perforation, intestinal obstruction, gangrene gut and those undergoing reanastomosis for anastomotic leakage. Except for this last group (p=0.030), none of the groups had a statistical association with the occurrence of leak. Some studies have underlined the importance of intestinal obstruction as a determinant of suture line disruption[13,22] although we were not able to arrive at similar conclusions. Hesp and co workers[33] had opined that reanastomoses were prone for subsequent releakage, and our analysis has validated their findings.

Widal serology in association with spontaneous small bowel perforations was used in our study for the purpose of diagnosing Typhoid fever. We noted 7 cases of typhoid perforation, the other 15 in whom the

test was negative being assigned to the group of non specific enteric perforations.

## Intraoperative factors

We noted that the mean duration of surgery in the leak group was significantly higher than in the control group (p-0.048). This is most probably reflective of the difficulties faced intraoperatively which might later predispose to leakage. Alves et al[27] found difficulties encountered during the constructions of large bowel anastomoses to be predictive of subsequent anastomotic dehiscence.

We found that the presence of peritoneal contamination had an association with the occurrence of anastomotic leakage (p=0.023). This factor was also found to be independently predictive of suture line dehiscence by investigators in 2 separate studies[22, 27].

The level of anastomotic construction had no bearing on the incidence of leak in our study. Hesp and co workers[33] remarked that jejunojejunal anastomoses were less prone to leakage that those constructed distally, but no significant differences were observed in this regard in their study. Golub[22], Pickleman[30] and colleagues had reported that they found no differences between end to end, end to side or side to side anastomoses in their studies. Alves et al[27] reported colocolic anastomoses as having a significant risk of leakage, but a comparison was not within the scope of our study.

Intraoperative hypotension (systolic blood pressure below 80mm Hg) was found to be an important predictor of anastomotic dehiscence by multivariate analysis in our series (p=0.044). The presence of this variable might explain the higher mean total fluid infused in the leak group. On the contrary, Golub and associates had noted that intraoperative hypotension (taken as less than 90mm Hg) was not that significant factor in their study (p=0.51).

Much has been said of the deleterious effects of peri operative blood transfusions, especially in terms of anastomotic integrity[20,21,22,27]. We analyzed the effects of intraoperative transfusion and found no relation between it and the occurrence of suture line disruption (p=0.25). The total fluid infused in the intraoperative period did however have a statistical association with the incidence of anastomotic dehiscence (p=0.037). This could be accounted for, as was mentioned before, by the development of oedema at the anastomotic region[45].

The usage of intra abdominal drains for patients undergoing primary anastomoses was found to have an association with suture line leak in our study (p-0.029). This supports the earlier views of some authors[27], yet contradicts the findings of others[22,30].

The grade of the operating surgeon did not have a bearing on the anastomotic outcome in our analysis. A senior had was always present during the surgery, either assisting a trainee or performing the

anastomoses himself. The results of others[13,22,40] support our findings, although it has been noted before that even senior surgeons may have a lack of consistency in producing secure anastomoses, the frequency of their dehiscences ranging from 0.5 to 30% in one study[40].

## Post operative factors

Our analysis revealed that mechanical ventilatory support in the post operative period was associated with anastomotic dehiscence (p=0.019). Tissue hypoperfusion and decreased oxygen tension at the anastomotic level are known to adversely affect its healing[16]. Golub and colleagues mentioned COPD as a predictor of leakage, and stated that the low tissue oxygen levels seen in COPD might be the causative factor of dehiscence. We feel that the use of post operative ventilatory support could be a post operative indicator of poor tissue oxygenation and hence its association with disruption. A significant association between the use of vasopressor support and leakage could not be demonstrated (p=0.118) in spite of the fact that vasopressor agents are known to compromise mesenteric blood flow, thus perhaps affecting anastomotic healing.

## Multivariate analysis

Forward stepwise logistic regression analysis was done using variables that were found to be significant following univariate analysis. This revealed that hypoalbuminemia, low serum sodium levels, presence of peritoneal contamination and the occurrence of intraoperative

hypotension were each independently predictive of the risk of subsequent small intestinal suture line disruption.

Our results are similar to the findings of other investigators[22,27] who used the same method of statistical analysis. Golub et al[22] analyzed both small and large bowel anastomoses and found 5 independent factors predictive of anastomotic leak viz., COPD, bowel obstruction, peritonitis, corticosteroid usage, blood transfusions > 2 units and a serum albumin <3g%. Alves and co workers[27] focused purely on large bowel anastomoses and noted that preoperative leucocytosis, intraoperative septic conditions, difficulty in constructing the anastomoses, colocolic anastomoses and the amount of blood transfused to be associated with leakage from the anastomotic site.

## Management of suture line disruptions

In the patients who died, all of whom belonged to the suture line disruption group, no significant difference was observed between those who were managed conservatively and those who had a surgical exteriorization of the leak site (p=1.00). Reanastomosis after an anastomotic leak was shown to have a significant association with mortality (p=0.041), 8 out of the 10 patients it was performed in having died. We strongly recommend that small bowel suture line disruption should be treated by exteriorization of the leak site whenever relaparotomy is done for the same.

## Mortality data

The overall mortality rate in our study was 17.7% (13/73 deaths). All the deaths occurred in the leak group. The mortality rate in the leak group was 52% (13/25 deaths). Hesp et al[33] noted deaths in 18% of their patients who sustained small bowel anastomotic leakage.

There was no association noted between age and mortality in our series. Several investigators have established age as a predictor of mortality in small bowel perforations[36,44] but this sub group in our series also did not have any significant association with the risk of death.

Neither sex group had any predilection for mortality in our study. Other investigators[22] of bowel anastomotic leakages have observed similar results. Meier and colleagues[34] noted that female sex was one of the many factors that were associated with risk of mortality in typhoid enteric perforation. However, other studies on the latter have not reached the same conclusions.

Although several studies have not a correlation between duration of symptoms and mortality in specific and non specific small bowel perforations[34,37,42,43,44], we found no such association in that sub group of our patients (96 ± 63.5 hours in those who died Vs. 68.43 ± 54.25 hours in those who survived; p=0.427). Chaikof[41] has had results similar to ours, noting survival which was independent of delay in diagnosis.

Low serum albumin levels were significantly associated with increased mortality (p=0.022), as was an ASA grade of 3 and above (p=0.005).

The duration of surgery differed significantly between survivors and non survivors (p=0.02). This difference persisted even when considering just those who underwent primary anastomoses. Other studies did not find such an association[22,30].

Intraoperative hypotension was shown to have a significant association with mortality (p=0.004). Ventilatory support was also shown to be significantly associated with the incidence of mortality (p=0.013) while vasopressor usage had no such relation. The relative contributions of these factors to anastomotic morbidity or mortality have not been noted before, to our knowledge. Small to large bowel anastomoses were found to have a significant association with mortality in our study. The reasons for the same are not clear to us presently.

# CONCLUSION AND SUMMARY

This study was conducted in our hospital between November 2011 to October 2012 in order to define factors contributing small intestinal suture line disruption, to identify factors causing mortality after small bowel anastomoses and to suggest measures which might lessen their impact. We also wished to propose the ideal line of management for patients who have had an anastomotic disruption.

The overall suture line disruption rate in our study was 38.55% (32 of 83 anastomoses). We encountered anastomotic leakage in 33.8% (23 of 68) of small to small bowel anastomoses. The corresponding figure for small bowel to large bowel anastomoses was 60% (9 of 15). Emergency cased made up the majority in our group (96.3%). All the 13 deaths in our study occurred in the suture line disruption group.

Factors found to have a significant association with suture line disruption by univariate analysis included ASA grade of 3 or above, low hemoglobin, low serum albumin, low preoperative serum sodium, higher mean duration of surgery, presence of gross peritoneal contamination, use of drains in primary anastomoses, higher mean amount of fluids infused intraoperatively, intraoperative hypotension and post operative ventilatory support.

Multivariate analysis using binary logistic regression revealed that **hypoalbuminemia, low preoperative serum sodium levels, presence of gross peritoneal contamination and intraoperative hypotension were all predictive of suture line disruption**.

Reanastomosis after anastomotic leak was shown to be significantly associated with releakage, and with mortality. So small bowel suture line disruption should be treated by exteriorization of the leak site whenever relaparotomy is done for the same

**Factors associated with mortality included low serum albumin, increasing duration of surgery, ASA grade of 3 or above, intraoperative hypotension, post operative ventilatory support, reanastomosis for leak and small to large bowel anastomoses.** All the deaths in our series occurred in the suture line disruption group.

# ANNEXURE-1

## BIBLIOGRAPHY


1. Bishop WJ. *The Early History of Surgery.* London : Robert Hale Limited; 1960
2. Meade RH Surgery of the small intestine. In: *An introduction to the history of general surgery.* Philadelphia : WB Saunders Company; 1968
3. Burch JM., Franciose RJ, Moore EE, Biffl WL, Offner PJ. Single-layer continuous versus two-Layer interrupted intestinal anastomosis: a prospective randomized trial. *Ann Surg* 2000*;* 231(6): 832-7
4. Goligher JC, Morris C, McAdam WAF, De Dombal FT, Johnston D. A controlled trial of inverting versus everting intestinal suture in clinical large bowel surgery. *Br J Surg* 1970; 57 : 817-22
5. Waninger J, Kauffmann GW, Shah IA, Farthmann EH. Influence of distance between interrupted sutures and the tension of sutures on the healing of experimental colonic anastomoses. *Am J Surg* 1992;163 : 319-23
6. Brooks DC, Zinner MJ. Surgery of the small and large bowel. In: Zinner MJ, Schwartz SI, Ellis H, editors. *Maingot's abdominal operations.* 10th ed. Connecticut: Appleton & Lange; 1997. p.1309-1359.

7. Chassin JL, Rifkind LM, Sussman B et al. The stapled gastrointestinal anastomosis: incidence of post operative complications compared with the sutured anastomosis. *Ann Surg* 1981; 116:14-16

8. Jonsson K, Jiborn H, Zederfeldt B. Breaking strength of small intestinal anastomosis. *Am J Surg* 1983; 145: 800-3

9. Jonsson K, Jiborn H, Zederfeldt B. Changes in the collagen content of the small intestinal wall following anastomosis. *Am J Surg* 1985; 150:315-7

10. Jonsson K, Jiborn H, Zederfeldt B. Collagen metabolism in small intestinal anastomosis. *Am J Surg* 1987; 154: 288-91

11. Peel ALG, Taylor EW. Proposed definitions for the audit of postoperative infection: a discussed paper. *Ann R Coll Surg Engl* 1991; 73:385-8

12. Bruce J, Krukowski ZH, Al-Khairy G., Russell EM, Park KGM. Systematic review of the definition and measurement of anastomotic leak after gastrointestinal surgery. *Br J Surg 2001; 88:1157-68*

13. Jex RK, van Heerden JA, Wolff BG, Ready RL, Ilstrup DM. Gastrointestinal anastomoses. Factors affecting early complications. *Ann Surg* 1987; 206: 138-41

14. Nahai F, Lamb JM, Havican RG, Stone HH. Factors involved in the disruption of intestinal anastomoses. *Am Surg* 1977; Jan: 45-51.

15. Ahrendt GM, Tantry US, Barbul A. Intra-abdominal sepsis impairs colonic reparative collagen synthesis. *Am J Surg* 1996; 171: 102-8

16. Sheridan WG, Lowndes RH, Young HL. Tissue oxygen tension as a predictor of colonic anastomotic healing. *Dis Col Rectum* 1987; 30 : 867-71

17. Irvin TT, Goligher JC. Aetiology of disruption of intestinal anastomoses. *Br J Surg* 1973; 60: 461-4

18. Stoop MJ, Dirksen R, Hendriks T. Advanced age alone does not suppress anastomotic healing in the intestine. *Surgery* 1996; 119: 15-19

19. Behrman SW, Bertken KA, Stefanacci HA, Parks SN. Breakdown of intestinal repair after laparotomy for trauma : incidence, risk factors and strategies for prevension. *J Trauma* 1998; 45: 227-33.

20. Tadros T, Wobbes T, Hendriks T. Blood transfusion impairs the healing of experimental intestinal anastomoses. *Ann Surg* 1992; 215: 276-81

21. Wobbes T, Bemelmans BLH, Kuypers JHC, Beerthuizen GIJM, Theeuwes AGM. Risk of postoperative septic complications after abdominal surgical treatment in relation to perioperative blood transfusion. *Surg Gynecol Obset* 1990; 171: 59-62

22. Golub R, Golub RW, Cantu R, Stein HD. A multivariate analysis of factors contributing to leakage of intestinal anastomoses. *J Am Coll Surg* 1997; 184: 364 – 372

23. Demptsy DT, Buzby GP, Mullen JL. Nutritional assessment in the seriously ill patient. *J Am Coll Nutr* 1983; 2: 15-22

24. Collins JP, McCarthy ID, Hill GL. Assessment of protein nutrition in surgical patients- the value of anthropometrics. *Am J Clin Nutr* 1979; 32:1527-30

25. Brenner U, Wolters U, Said S, Muller JM. Anthropometric parameters for preoperative screening to predict high risk patients. *Acta Chir Belg* 1989; 89:19-24.

26. Detsky AS, Baken JP, O'Rourke K, Johnston N, Whitwell J, Mendelson RA et al. Predicting nutrition associated complications for patients undergoing gastrointestinal surgery. *J Parenter Enteral Nutr* 1987; 11:440-6

27. Alves A, Panis Y, Trancart D, Regmbeau JM, Pocard M, Valleur P. Factors associated with clinically significant anastomotic leakage after large bowel resection: multivariate analysis of 707 patients. *World Surg* 2002; 26: 499-502.

28. Gibbs J, Cull W, Henderson W, Daley J Hur K, Khuri SF. Preoperative serum albumin as a predictor of operative mortality and morbidity: results from the National VA Surgical Risk Study. *Arch Surg* 1999; 134:36-42.

29. Kudsk KA, Tolley EA, DeWitt RC, Janu PG, Blackwell AP, Yeary S et al. Preoperative albumin and surgical site identify surgical risk for major postoperative complications. *J Parenter Enteral Nutr* 2003; 27: 1-9

30. Pickleman J, Watson W, Cunningham J, Fisher SG, Gamelli R. The failed gastrointestinal anastomosis: an inevitable catastrophe? *J Am Coll Surg* 1999; 188: 473-482

31. Buchmiller- Crair TN, Kim CS, Won NH, Chopourian HL, Shaw KS, Fonkalsrud EW. Effects of acute anemia on the healing of intestinal anastomoses in the rabbit. *J Trauma* 2001; 51: 363-8

32. Irvin TT. Effects of malnutrition and hyperalimentation on wound healing. *Surg Gynecol Obste* 1978; 146: 33-7

33. Hesp WLEM, Lubbers EJC, de Boer HHM, Hendriks T. Anastomotic insufficiency in small bowel surgery – incidence and treatment. *Langenbecks Arch Chir* 1986; 368: 105-11

34. Meier DE, Imediegwu OO, Tarpley JL. Perforated typhoid enteritis: operative experience with 108 cases. *Am J Surg* 1989; 157423-7

35. Santillana M. Surgical complications of typhod fever: enteric perforation. *World J Surg* 1991; 15: 170-5

36. Mock CN. Amaral J, Visser LE. Improvement in survival from typhoid ileal perforation. *Ann Surg* 1992; 215: 244-9

37. Chatterjee J, Jagdish S, Pai D, Satish N, Jayadev D, Reddy PS. Changing trends of typhoid ileal perforations over three decades in Pondicherry. *Trop Gastroenterol* 2001; 22: 155-8

38. Fawcett A, Shembekar M, Church JS, Vashisht R, Springall RG, Nott DM. Smoking, hypertension and colonic anastomotic healing; a combined clinical and histopathological study. *Gut* 1996; 38: 714 – 8

39. Colin JF, Elliot P, Ellis H. The effect of uraemia upon would healing: an experimental study. *Br J Surg* 1979; 66: 793-7

40. Fielding LP, Stewart-Brown S, Blesovsky L, Kearney G. Anastomotic integrity after operations for large bowel cancer; a multicentre study. *BMJ* 1980; 2: 411-4.

41. ChaikofEL. Non traumatic perforation of the small bowel. *Am J Surg 1987; 153: 355-8*

42. Chatterjee H, Pai D, Jagdish S, Satish N, Jayadev D, Reddy PS. The pattern of non typhoid ileal perforation over three decades in Pondicherry (Submitted)

43. Eggleston FC, Santoshi B, Singh CM. Typhoid perforation of the bowel. *Ann Surg* 1979; 190: 31-5

44. Akgun Y, Bac B, Boylu S, Aban N, Tacyildiz I. Typhoid enteric perforation. *Br J Surg* 1995; 82: 1512-5

45. Chan STF, Kapadia CR, Johnson AW, Radcliffe AG, Dudley HAF. Extracellular fluid volume expansion and third space sequestration at the site of small bowel anastomoses. *Br J Surg* 1983; 70: 36-9.

## ANNEXURE-2

## CLINICAL PROFORMA

NAME:

AGE:

SEX:

IP.NO

OCCUPATION:

ADDRESS:

**CLINICAL HISTORY**

DURATION OF SYMPTOMS(IN EMERGENCY CASES)

- ABDOMINAL PAIN
- ABDOMINAL DISTENSION
- VOMITING
- FEVER

PERSONAL HISTORY

- TOBACCO,ALCOHOL,STEROID MEDICATIONS

PAST HISTORY

- DIABETES MELLITUS
- HYPERTENSION
- COPD

**GENERAL EXAMINATION**

- PALLOR
- PULSE RATE
- BLOOD PRESSURE
- RESPIRATORY RATE
- NUTRITIONAL STATUS (WEIGHT,HEIGHT,MID ARM CIRCUMFERANCE,TRICEPS FOLD THICKNESS)
- TEMPERATURE

**PER ABDOMEN**

- TENDERNESS
- DISTENSION
- GUARDING/RIGIDITY
- BOWEL SOUNDS

**PRE OPERATIVE DIAGNOSIS**

**INVESTIGATIONS**

- HB
- SERUM TOTAL PROTEIN/ALBUMIN
- UREA/CREATININE
- ELECTROLYTES
- BLOOD CULTURE
- WIDAL
- RADIOLOGICAL INVESTIGATIONS(X-RAY,USG ABDOMEN,CT ABDOMEN)

**SURGERY DONE**

- ILEOILEAL ANASTOMOSIS
- ILEOCOLIC ANASTOMOSIS
- JEJUNOJEJUNAL ANASTOMOSIS
- JEJUNOCOLIC ANASTOMOSIS
- END TO END/END TO SIDE
- SINGLE LAYER/DOUBLE LAYER

**INTRA OP DETAILS**

- DURATION OF SURGERY
- PRESENCE OF GROSS PERITONEAL CONTAMINATION
- GANGRENE OF BOWEL
- NATURE OF PERITONEAL CONTAMINANT
- SITE OF PATHOLOGY IN SMALL BOWEL
- PRESENCE,NUMBER,SIZE OF PERFORATIONS
- SUTURE MATERIAL USED
- PRESENCE OF ANY DISTAL OBSTRUCTION
- AMOUNT AND NATURE OF INTRAOP FLUIDS GIVEN
- INTRA OP HEMODYNAMIC EVENT
- USE OF DRAINS
- OPERATING SURGEON

**POST OP DETAILS**

- VASOPRESSOR SUPPORT
- VENTILATORY SUPPORT
- ANTIBIOTICS USED
- USE OF STEROIDS
- PRESENCE OF WOUND INFECTION
- STARTING ORAL FLUIDS

## ANNEXURE-3

## KEY TO MASTER CHART

M - Male

F - Female

\+ - Present

\- - Absent

NS - Non Smoker

S - Smoker

A - Alcoholic

NA - Non Alcoholic

P - Perforation

IO - Intestinal Obstruction

G - Gangrene

F - Faeces

B - Bile

II - Ileoileal

IC - Ileocolic

JJ - Jejunojejunal

JC - Jejunocolic

JS - Junior surgeon

SS - Senior surgeon

E-E - End to End anastomosis

E-S - End to Side anastomosis

WR - Wedge resection

A - Alive

D - Dead

# ANNEXURE-4

## MASTER CHART

## CASES

| Sl. No. | Name | IP. No. | Age | Sex | Duration of Symptoms (Hrs) | Smoking | Alcohol Intake | Pulse Rate | Blood Pressure (Systolic mm/hg) | Respiratory Rate (Rate/min) | Mid arm circumference (cm) | Triceps Fold Thickness (mm) | Body mass Index | Hb (gm/dl) | Total Protein (g/dl) | Albumin (g/dl) | Na Level (mEq/l) | K level (mEq/l) | Urea (mg/dl) | Creatinine (mg/dl) | Indications for Surgery | WIDAL | Duration of Surgery (mins) | Presence of Gross Contamination | Nature of Peritoneal Contaminant | Patients with Multiple perforation | Distance from IC Junction (cm) | Distance from DJ Junction (cm) | Anastomotic Procedure | Nature of Anastomosis | Amount of Fluid Infused (ml) | Intraoperative hypotension | Use of drains | Surgeon | Ventilatory Support | Vaso pressor support | Time of Starting Orals (hrs) | Outcome |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | SELVAM | 024456 | 41 | M | 72 | NS | NA | 80 | 100 | 20 | 26 | 7 | 21 | 9 | 5.8 | 2.3 | 135 | 3.6 | 20 | 1 | P | - | 120 | + | F | - | 10 | | II | E-E | 2000 | - | + | JS | - | - | 120 | A |
| 2 | LAKSHMI | 300956 | 46 | F | 72 | NS | NA | 100 | 110 | 20 | 20 | 8 | 17 | 10.6 | 6.2 | 2.7 | 130 | 4.5 | 50 | 1.1 | IO | - | 120 | - | | - | 50 | | II | E-E | 2000 | - | - | SS | - | - | 120 | A |
| 3 | CHELLAIAH | 38819 | 26 | M | 96 | S | A | 105 | 100 | 15 | 22 | 6 | 23 | 9 | 6 | 2.6 | 127 | 5 | 50 | 1.2 | P | + | 120 | + | F | | 20 | | II | W-R | 2500 | - | + | SS | - | - | 120 | A |
| 4 | THIRUPPATHI | 40934 | 33 | M | 118 | NS | NA | 110 | 80 | 18 | 25 | 8 | 21 | 9.6 | 5.2 | 3 | 137 | 3.6 | 50 | 0.5 | IO | - | 150 | - | | - | 40 | | II, IC | E-S | 3500 | - | + | SS | + | - | 120 | D |
| 5 | PANDIAMMAL | 38878 | 70 | F | 72 | NS | NA | 90 | 80 | 20 | 27 | 6 | 18 | 7.5 | 5.4 | 2.9 | 125 | 4.4 | 50 | 0.5 | G | - | 150 | + | F | - | 20 | | IC | E-E | 4000 | + | + | JS | + | + | 144 | D |
| 6 | PALANISAMY | 38895 | 32 | M | 96 | S | A | 85 | 110 | 20 | 21 | 9 | 22 | 9.6 | 5.8 | 2.5 | 130 | 4 | 25 | 0.9 | IO | - | 120 | + | | - | 30 | | II | E-E | 3000 | - | + | SS | - | - | 120 | A |

| | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 7 | MUTHU | 34988 | 74 | F | 120 | NS | NA | 95 | 90 | 18 | 25 | 7 | 18 | 10 | 4.3 | 2.4 | 129 | 3.7 | 30 | 2 | P | - | 120 | + | F | - | 10 | | II, JJ | E-E | 3500 | + | + | SS | + | + | 120 | D |
| 8 | MUTHURAJA | 32899 | 45 | M | 72 | S | A | 100 | 90 | 15 | 21 | 6 | 18 | 6 | 4.6 | 2.4 | 125 | 4.4 | 40 | 0.6 | G | - | 180 | + | B | + | | 40 | JC | E-S | 4000 | - | + | JS | - | - | 144 | D |
| 9 | KASTHURI | 38853 | 60 | F | 72 | NS | NA | 80 | 90 | 30 | 25 | 8 | 19 | 9 | 5.6 | 2.6 | 133 | 3.8 | 55 | 0.6 | P | - | 210 | + | F | - | 60 | | II, IC | E-S | 4000 | + | + | SS | + | + | 120 | D |
| 10 | JEGATHEESH | 40950 | 28 | M | 120 | NS | NA | 95 | 120 | 20 | 25 | 6 | 21 | 8 | 4.6 | 2.8 | 135 | 4.5 | 50 | 0.7 | P | - | 90 | + | B | + | 30 | | IC | E-E | 2000 | - | + | SS | - | - | 120 | A |
| 11 | RANIAMMAL | 36986 | 74 | F | 120 | NS | NA | 90 | 110 | 18 | 22 | 6 | 22 | 9 | 4.4 | 2 | 126 | 4.3 | 45 | 0.8 | IO | - | 150 | + | | - | 20 | | II | E-E | 3500 | - | + | JS | - | - | 120 | D |
| 12 | MUGESKUMAR | 04084 | 51 | M | 72 | S | A | 88 | 100 | 20 | 28 | 7 | 20 | 7.4 | 5.6 | 2.5 | 131 | 5 | 30 | 0.7 | P | + | 120 | + | B | - | 40 | | II | W-R | 3000 | - | + | SS | - | - | 120 | A |
| 13 | MUTHAIAH | 35143 | 29 | M | 120 | NS | NA | 90 | 90 | 30 | 25 | 8 | 26 | 8.6 | 5.4 | 2.7 | 132 | 3.6 | 20 | 0.6 | P | - | 150 | + | F | - | 30 | | II, IC | E-S | 4000 | + | + | SS | + | + | 120 | D |
| 14 | VELUMANI | 040828 | 44 | F | 96 | NS | NA | 95 | 110 | 20 | 20 | 7 | 23 | 9 | 5.4 | 2.8 | 136 | 4.3 | 50 | 1.2 | IO | - | 180 | + | | + | 40 | | II | E-E | 2000 | - | + | JS | - | - | 144 | A |
| 15 | PAULSAMY | 040847 | 35 | M | 72 | S | NA | 90 | 100 | 15 | 25 | 9 | 20 | 11.6 | 4.4 | 2.6 | 140 | 3.9 | 54 | 1 | IO | - | 180 | + | | - | 50 | | IC | E-S | 3500 | - | - | SS | - | - | 120 | D |
| 16 | NALLIAPPAN | 042818 | 39 | M | 96 | NS | NA | 110 | 90 | 18 | 23 | 7 | 20 | 10 | 5 | 2.9 | 129 | 4.2 | 50 | 0.8 | P | - | 210 | + | B | - | 40 | | II, JJ | E-E | 2000 | - | + | SS | + | - | 120 | A |
| 17 | MEENAKSHI | 38924 | 50 | F | 72 | NS | NA | 90 | 80 | 25 | 27 | 8 | 19 | 8 | 5.5 | 2.7 | 110 | 3.7 | 35 | 1.1 | P | + | 90 | + | F | + | 30 | | II | E-E | 4500 | + | + | JS | + | + | 120 | D |
| 18 | MUTHU | 040415 | 47 | M | 72 | S | A | 95 | 110 | 30 | 24 | 6 | 21 | 9 | 4.8 | 2 | 130 | 4 | 70 | 3 | G | - | 180 | + | B | + | | 30 | JC | E-S | 3000 | + | - | JS | + | + | 144 | D |
| 19 | AYYAVU | 302096 | 30 | M | 120 | NS | NA | 110 | 120 | 20 | 29 | 6 | 24 | 8 | 4.8 | 3 | 130 | 3.8 | 45 | 0.7 | P | - | 150 | + | F | - | 50 | | II, JJ | E-E | 3500 | - | + | SS | - | - | 144 | A |
| 20 | CHINNAPPAN | 042789 | 48 | M | 120 | NS | NA | 85 | 100 | 15 | 23 | 9 | 21 | 8.4 | 6 | 3 | 128 | 3.9 | 45 | 1 | IO | - | 150 | + | | + | 60 | | II | E-E | 2500 | - | - | SS | - | - | 120 | A |

| | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 21 | JAYA | 042508 | 34 | F | 72 | NS | NA | 95 | 90 | 25 | 25 | 6 | 22 | 5 | 6.2 | 2.5 | 137 | 3.9 | 70 | 0.9 | G | - | 180 | + | F | - | 10 | | IC | E-S | 4000 | + | + | JS | + | + | 168 | D |
| 22 | PURSOTHAMMAN | 44769 | 31 | M | 120 | S | A | 95 | 110 | 20 | 30 | 7 | 23 | 9.4 | 4.2 | 2.7 | 131 | 4.1 | 55 | 0.9 | P | - | 90 | + | B | - | 50 | | II, JJ | E-E | 3000 | - | + | SS | - | - | 120 | A |
| 23 | RAMAR | 43750 | 49 | M | 72 | S | NA | 100 | 80 | 25 | 26 | 8 | 24 | 10 | 5.2 | 3.1 | 131 | 4.1 | 55 | 3 | G | - | 150 | + | F | + | 60 | | II | E-E | 4000 | + | + | JS | + | + | 168 | D |
| 24 | BOSE | 44736 | 36 | M | 72 | NS | NA | 100 | 120 | 15 | 24 | 8 | 24 | 8 | 5 | 2.7 | 134 | 4.2 | 55 | 1.1 | IO | - | 150 | - | | - | 10 | | II | E-E | 3000 | - | + | JS | - | - | 120 | A |
| 25 | RAJENDRAN | 37083 | 50 | M | 120 | NS | NA | 100 | 120 | 20 | 28 | 7 | 21 | 11 | 5.5 | 2.6 | 130 | 3.9 | 50 | 0.8 | IO | - | 150 | + | | - | 20 | | II | E-E | 2000 | - | + | SS | - | - | 120 | D |
| **CONTROLS** | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| 26 | PERIYA KARUPPAN | 47814 | 40 | M | 48 | S | NA | 75 | 90 | 25 | 20 | 8 | 22 | 6 | 6.6 | 3.4 | 140 | 4.4 | 30 | 0.5 | P | - | 180 | + | F | - | 10 | | II | W-R | 1500 | - | + | JS | - | - | 120 | |
| 27 | SUNDARI | 54535 | 42 | F | 12 | NS | NA | 90 | 130 | 18 | 22 | 6 | 17 | 10 | 6.2 | 3.5 | 138 | 4.2 | 45 | 1.1 | IO | - | 90 | + | | - | 40 | | II | E-E | 2000 | - | + | SS | - | - | 120 | |
| 28 | MOOKAN | 29920 | 23 | M | 96 | NS | NA | 70 | 100 | 20 | 25 | 7 | 20 | 8.6 | 6 | 2.7 | 133 | 4.1 | 60 | 0.5 | P | - | 90 | - | B | - | 20 | | II | E-E | 2000 | - | - | SS | - | - | 120 | |
| 29 | PALANIAMMAL | 48677 | 39 | F | 72 | NS | NA | 90 | 120 | 18 | 24 | 7 | 18 | 5 | 6 | 2.8 | 131 | 3.6 | 70 | 1.2 | G | - | 120 | + | F | - | 20 | | II, JJ | E-E | 3500 | - | + | SS | + | + | 144 | |
| 30 | AYYADURAI | 45213 | 30 | M | 48 | S | A | 80 | 80 | 30 | 26 | 6 | 17 | 10 | 5.2 | 3.3 | 136 | 4 | 55 | 0.5 | IO | - | 180 | - | | + | 20 | | II | E-E | 2500 | - | - | SS | - | - | 96 | |
| 31 | RAMAR | 46761 | 41 | M | 120 | S | NA | 80 | 120 | 20 | 30 | 7 | 26 | 11 | 6 | 3.8 | 135 | 4.3 | 40 | 1.2 | P | - | 120 | + | B | - | | 20 | JJ | W-R | 3000 | - | + | JS | - | - | 120 | |
| 32 | DHANALAKSHMI | 50213 | 23 | F | 48 | NS | NA | 90 | 80 | 30 | 24 | 8 | 18 | 12 | 5.8 | 3 | 140 | 3.9 | 50 | 0.6 | G | - | 150 | - | B | - | 10 | | IC | E-E | 3500 | - | - | JS | - | - | 120 | |
| 33 | PERIYASAMY | 46087 | 33 | M | 48 | NS | NA | 70 | 90 | 30 | 25 | 6 | 21 | 10 | 5.3 | 2.7 | 136 | 3.7 | 35 | 1.1 | P | + | 150 | + | F | - | 30 | | II | W-R | 2000 | - | - | SS | - | - | 120 | |

| | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 34 | PANDIAMMAL | 49027 | 25 | F | 120 | NS | NA | 95 | 100 | 20 | 24 | 10 | 19 | 10.4 | 6.5 | 3.2 | 135 | 3.6 | 40 | 1.5 | G | - | 180 | + | F | - | 30 | | II, JJ | E-E | 2000 | + | + | SS | + | + | 149 | |
| 35 | KRISHNARAJ | 50535 | 39 | M | 120 | S | A | 80 | 80 | 15 | 25 | 7 | 20 | 9.4 | 5 | 2.5 | 136 | 4.6 | 20 | 1 | IO | - | 180 | + | | + | 10 | | II | E-E | 1500 | - | + | SS | - | - | 96 | |
| 36 | RAJU | 46676 | 13 | M | 6 | NS | NA | 100 | 110 | 18 | 21 | 6 | 17 | 9 | 4.8 | 2.6 | 135 | 3.8 | 45 | 0.6 | IO | - | 150 | + | | - | | 30 | JJ | E-E | 2500 | - | + | JS | - | - | 96 | |
| 37 | KURIAMMAL | 50668 | 18 | F | 96 | NS | NA | 85 | 90 | 30 | 23 | 8 | 23 | 9 | 6.4 | 3.7 | 130 | 3.7 | 60 | 1.5 | G | - | 150 | - | B | - | 40 | | IC | E-E | 1500 | - | - | SS | - | - | 120 | |
| 38 | KATHIRESAN | 50655 | 35 | M | 24 | S | NA | 70 | 120 | 15 | 29 | 6 | 20 | 8.6 | 4.9 | 2.5 | 133 | 3.8 | 40 | 0.9 | P | - | 180 | + | F | - | | 50 | JJ | W-R | 1500 | - | + | SS | - | - | 120 | |
| 39 | MUNIASAMY | 48735 | 23 | M | 48 | S | A | 95 | 80 | 25 | 24 | 7 | 22 | 9 | 4.8 | 2 | 131 | 4 | 55 | 0.7 | P | + | 120 | + | B | - | 50 | | II | E-E | 2500 | - | - | JS | - | - | 120 | |
| 40 | THANGAMMAL | 29952 | 44 | F | 72 | NS | NA | 75 | 100 | 18 | 23 | 7 | 19 | 9.4 | 6.8 | 3.1 | 133 | 4.1 | 70 | 1.4 | G | - | 120 | + | F | - | 10 | | II, JJ | E-E | 2500 | - | + | SS | + | + | 168 | |
| 41 | KARUPPAIAH | 48672 | 24 | M | 45 | S | NA | 70 | 100 | 20 | 22 | 8 | 18 | 6 | 6.5 | 3 | 140 | 3.9 | 25 | 0.8 | G | - | 180 | + | F | + | 60 | | IC | E-E | 2000 | - | + | SS | - | - | 144 | |
| 42 | VANAJA | 50588 | 36 | F | 72 | NS | NA | 95 | 80 | 25 | 27 | 8 | 19 | 10.2 | 5.6 | 2.6 | 134 | 4 | 50 | 0.8 | P | - | 90 | - | B | - | | 40 | JJ | E-E | 3500 | - | - | JS | - | - | 120 | |
| 43 | SARAVANAN | 50624 | 23 | M | 72 | S | A | 90 | 100 | 18 | 24 | 10 | 21 | 10 | 5.1 | 3.6 | 132 | 4.3 | 40 | 0.6 | P | - | 90 | + | F | - | 70 | | II | E-E | 3000 | - | + | SS | - | - | 120 | |
| 44 | ESWARI | 50627 | 43 | F | 12 | NS | NA | 70 | 90 | 20 | 23 | 9 | 19 | 11 | 7 | 2.4 | 130 | 4.2 | 45 | 0.9 | IO | - | 190 | + | | - | 20 | | IC | E-S | 2500 | - | + | SS | - | - | 96 | |
| 45 | SELVAM | 50592 | 31 | M | 72 | S | NA | 100 | 100 | 15 | 23 | 6 | 20 | 10 | 6.8 | 3.9 | 132 | 4.1 | 30 | 0.7 | P | + | 90 | - | P | - | 30 | | II | E-E | 3000 | - | - | JS | - | - | 120 | |
| 46 | ANNAMAYEL | 48761 | 49 | F | 72 | NS | NA | 100 | 80 | 18 | 24 | 6 | 22 | 10.4 | 6 | 3.2 | 137 | 4.6 | 65 | 0.7 | IO | - | 120 | - | | - | | 30 | JJ | E-E | 3500 | - | - | SS | - | - | 120 | |
| 47 | NATCHIMUTHU | 48671 | 32 | M | 12 | S | A | 80 | 100 | 18 | 28 | 7 | 23 | 9 | 5.4 | 2.8 | 140 | 3.9 | 50 | 0.8 | IO | - | 150 | + | | - | | 40 | JJ | E-E | 2500 | - | + | JS | - | - | 120 | |

| | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 48 | ANITHA DEVI | 51532 | 48 | F | 72 | NS | NA | 90 | 90 | 30 | 24 | 9 | 21 | 12 | 5.6 | 3.9 | 130 | 4.2 | 45 | 1 | P | + | 120 | - | B | + | 30 | | II | W-R | 2000 | - | - | JS | - | - | 144 | |
| 49 | PERUMAL SMAY | 50673 | 33 | M | 72 | NS | NA | 75 | 80 | 18 | 22 | 8 | 21 | 5 | 5.8 | 2.7 | 138 | 3.7 | 45 | 1.3 | G | - | 90 | + | F | - | 40 | | IC | E-S | 3000 | - | + | SS | - | - | 96 | |
| 50 | DEEPA | 52541 | 58 | F | 72 | NS | NA | 95 | 130 | 20 | 24 | 7 | 21 | 10.4 | 5.6 | 2.9 | 143 | 4.4 | 55 | 0.9 | P | - | 180 | - | P | - | | 20 | JJ | E-E | 2500 | - | - | JS | - | - | 120 | |
| 51 | RAMU | 50670 | 26 | M | 48 | S | A | 105 | 110 | 18 | 29 | 6 | 17 | 11.6 | 5.7 | 2.9 | 136 | 3.8 | 75 | 1.5 | G | - | 120 | + | F | - | 50 | | II | E-E | 3500 | - | + | JS | + | + | 120 | |
| 52 | MANOHARAN | 50593 | 59 | M | 72 | NS | NA | 75 | 130 | 18 | 26 | 7 | 18 | 12.4 | 6.3 | 4 | 139 | 4.5 | 50 | 1 | P | - | 140 | + | B | - | | 50 | JJ | E-E | 1500 | - | + | SS | - | - | 120 | |
| 53 | PANDIAMMAL | 50602 | 23 | M | 48 | NS | NA | 105 | 110 | 18 | 30 | 8 | 20 | 10 | 5.6 | 3 | 140 | 4.5 | 55 | 0.6 | G | - | 150 | - | F | + | 60 | | IC | E-S | 2000 | - | - | JS | - | - | 144 | |
| 54 | RAMUTHAI | 48695 | 60 | F | 12 | NS | NA | 110 | 130 | 20 | 24 | 8 | 22 | 10 | 5.9 | 2.5 | 134 | 3.6 | 50 | 0.7 | IO | - | 180 | + | | - | 70 | | II | E-E | 3000 | - | - | JS | - | - | 120 | |
| 55 | SRINIVASAN | 51527 | 53 | M | 48 | NS | NA | 110 | 110 | 18 | 25 | 10 | 21 | 9.4 | 6.8 | 3 | 140 | 4.1 | 30 | 0.5 | IO | - | 150 | - | | - | | 50 | JJ | E-E | 1500 | - | - | SS | - | - | 120 | |
| 56 | VANCHU | 054428 | 24 | F | 120 | NS | NA | 85 | 110 | 15 | 28 | 7 | 21 | 9 | 6 | 3.2 | 136 | 4.5 | 50 | 0.5 | IO | - | 90 | + | B | - | 10 | | II | E-E | 1500 | - | + | JS | - | - | 120 | |
| 57 | KARUPPUSAMY | 052612 | 28 | M | 72 | S | A | 100 | 90 | 25 | 30 | 6 | 25 | 6 | 5.1 | 3.3 | 130 | 3.8 | 50 | 1 | P | - | 150 | - | F | - | | 30 | JJ | E-E | 2000 | - | - | SS | - | - | 144 | |
| 58 | SIVASUBRAMANI | 050683 | 47 | M | 48 | NS | NA | 80 | 100 | 18 | 23 | 9 | 17 | 11 | 6.5 | 3.1 | 138 | 3.6 | 55 | 0.5 | G | - | 120 | + | | - | 20 | | II | E-E | 1500 | - | + | SS | + | + | 120 | |
| 59 | MUNIAMMAL | 056733 | 24 | F | 120 | NS | NA | 90 | 120 | 15 | 22 | 6 | 20 | 10 | 5 | 2.7 | 131 | 4.3 | 63 | 0.6 | IO | - | 180 | + | | - | | 20 | JJ | E-E | 2000 | - | - | JS | - | - | 96 | |
| 60 | MOHAN | 050672 | 52 | M | 72 | S | A | 100 | 100 | 18 | 24 | 7 | 19 | 9 | 6.2 | 3 | 139 | 3.7 | 35 | 0.8 | IO | - | 150 | - | | - | 40 | | II | E-E | 2500 | - | + | SS | - | - | 120 | |
| 61 | KASIMURUGAN | 054507 | 46 | M | 48 | S | NA | 85 | 110 | 15 | 22 | 8 | 17 | 9.4 | 5.7 | 3 | 135 | 3.7 | 35 | 0.6 | P | - | 120 | - | B | - | | 40 | JJ | E-E | 2000 | - | - | JS | - | - | 120 | |

| | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 62 | JEGAN | 051343 | 57 | M | 48 | NS | NA | 95 | 110 | 20 | 27 | 8 | 22 | 11 | 5.4 | 3.6 | 140 | 3.8 | 58 | 0.6 | IO | - | 180 | + | | - | 30 | | II | E-E | 2500 | - | - | SS | - | - | 120 | |
| 63 | KARTHIKA | 056737 | 24 | F | 72 | NS | NA | 75 | 110 | 18 | 27 | 6 | 23 | 10.4 | 4.8 | 2.6 | 136 | 4 | 40 | 0.7 | G | - | 150 | - | B | - | 10 | | IC | E-E | 3000 | - | + | JS | - | - | 144 | |
| 64 | MAYILRAJ | 051516 | 56 | M | 48 | S | A | 105 | 80 | 30 | 24 | 7 | 26 | 11 | 5.2 | 2.9 | 132 | 3.6 | 40 | 1.1 | IO | - | 120 | + | | - | 50 | | II | E-E | 3000 | - | + | SS | - | - | 120 | |
| 65 | MURUGAN | 052574 | 51 | M | 72 | NS | NA | 90 | 120 | 20 | 26 | 8 | 21 | 7 | 4 | 2.5 | 135 | 4.2 | 45 | 0.7 | IO | - | 90 | + | | - | 10 | | II | E-E | 2000 | - | + | SS | - | - | 120 | |
| 66 | SAGUNTHALA | 052655 | 27 | F | 120 | NS | NA | 95 | 110 | 18 | 21 | 7 | 18 | 11 | 6 | 3.2 | 136 | 4 | 52 | 0.9 | IO | - | 120 | - | | - | 70 | | II | E-E | 2500 | - | + | SS | - | - | 120 | |
| 67 | PRABHAKARAN | 052516 | 55 | M | 72 | S | A | 105 | 90 | 20 | 26 | 7 | 19 | 10 | 5 | 3.4 | 134 | 3.9 | 45 | 0.9 | IO | - | 90 | - | | - | 20 | | II | E-E | 3500 | - | + | SS | - | - | 120 | |
| 68 | VIJAYAKUMRA | 054568 | 45 | M | 120 | NS | NA | 80 | 120 | 15 | 24 | 7 | 18 | 10 | 5.3 | 2.8 | 133 | 3.9 | 60 | 0.7 | P | - | 90 | - | F | - | 60 | | II | E-E | 1500 | - | + | SS | - | - | 96 | |
| 69 | RAMAIAH | 052649 | 61 | M | 72 | NS | NA | 95 | 110 | 18 | 27 | 7 | 20 | 9 | 6 | 4 | 136 | 4.4 | 45 | 0.8 | IO | - | 150 | - | | - | 70 | | II | E-E | 3000 | - | + | SS | - | - | 120 | |
| 70 | PAPPAIYA | 054454 | 54 | M | 120 | S | NA | 100 | 90 | 25 | 29 | 8 | 22 | 10.4 | 5.5 | 2.9 | 134 | 4.6 | 30 | 0.9 | IO | - | 180 | - | | - | 70 | | II | E-E | 2500 | - | + | SS | - | - | 120 | |
| 71 | MANIKANDAN | 056996 | 62 | M | 72 | NS | NA | 85 | 120 | 18 | 27 | 8 | 21 | 11 | 5.8 | 3.1 | 134 | 4.1 | 65 | 1 | IO | - | 120 | - | | - | 60 | | II | E-E | 2500 | - | + | SS | - | - | 120 | |
| 72 | MURUGAN | 056760 | 29 | M | 120 | NS | NA | 105 | 110 | 18 | 28 | 6 | 23 | 10.4 | 5.8 | 3.8 | 135 | 4.2 | 30 | 1.2 | IO | - | 90 | - | | - | 40 | | II | E-E | 3000 | - | + | SS | - | - | 96 | |
| 73 | BABU | 056134 | 50 | M | 72 | NS | NA | 95 | 120 | 25 | 21 | 8 | 17 | 10 | 5.7 | 2.8 | 137 | 4.3 | 70 | 0.8 | IO | - | 180 | - | | - | 30 | | II | E-E | 2500 | - | + | SS | - | - | 120 | |